DISSERTATION ON
AETIOPATHOLOGICAL STUDY OF HOARSENESS OF VOICE

Submitted in partial fulfillment of the requirement for the award of the degree of M.S. Branch IV

(Otorhinolaryngology)

Department of Otorhinolaryngology

Tirunelveli Medical College

Tirunelveli

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
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This is to certify that this dissertation on “Aetiopathological study of hoarseness of voice” presented here in by Dr. Chitra S., is the original work done in the Department of Otorhinolaryngology, Govt. Tirunelveli Medical College hospital, Tirunelveli in partial fulfillment of regulations of the Tamilnadu Dr. M.G.R. Medical University, Chennai for the award of M.S. (otorhinolaryngology) under guidance and supervision during the academic year 2010-2013.

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Declaration

I Dr. Chitra S. Solemnly declare that this dissertation, titled “Aetiopathological study of hoarseness of voice” is a bonafide record of work done by me in the department of Otorhinolaryngology, Govt. Tirunelveli Medical College, Tirunelveli Under the guidance of Dr. S. Sureshkumar, M.S D.LO Prof & HOD of Otorhinolaryngology, Govt.Tirunelveli Medical College, Tirunelveli

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This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr. **S.CHITRA**, *a* **MS POSTGRADUATE IN ENT** *in the Department of* **ENT**, *of Tirunelveli Medical College Hospital, Tirunelveli titled* **"ETIOPATHOLOGICAL STUDY OF HOARSENESS OF VOICE"** *registered by the* IEC *as* **050/ENT**/IEC/2011 *dated.* **25.02.2011**. *The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.*

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TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

Voice is the primary means of communication for human both socially and in the work place[1]. Human voice serves number of communicative functions, some associated with this spoken language and others unrelated to speech and language voice alone, can communicate several nonverbal messages[2]. Clear pleasing confident voice convey positive impression of adequate personality while weak self effacing voice that necessitates the listener straining to hear reflect shy – withdrawn personality[3]. Voice disorders are rarely life threatening, but cause tremendous alteration in day today living and should not be underestimated as a medical disorder[5]. First persons to be approached when voice sounds abnormal is usually otolaryngologists[6].

Hoarseness is a symptom, not a diagnosis. It is a common symptom encountered by primary care providers. Hoarseness has a lifetime prevalence of 30%[1]. At any point in time, 3% to 9% of general population is affected by some type of voice abnormality [7,8] the negative effect of voice disturbance on quality of life and economic productivity is significant and has been well documented[8,9,10]. Hoarseness is the term used to described a change in normal voice. Quality and it is invariability the earliest manifestation of a large variety of conditions directly and indirectly affecting the voice apparatus [1]

Jackson & Jackson (1930) feel that hoarseness is the most important symptom of laryngeal disease and it is only absent when the cords and the motor mechanism are entirely free from disease [11]. The disease can result from a spectrum of conditions ranging from the common cold to a malignancy.

Hoarseness refers to a disturbance in voice, hoarseness is used by

patients to describe a breathy quality to voice, a roughness, a pitch change or sometimes some degree of dysarthria. Voice refers to sound produced by vibrating vocal folds. A voice problem traces its roots to the phonology mechanism at the vocal fold level. Speech refers to the train of sounds shaped by the phaynx, tongue, Oral cavity and lips to from words and phrases.

Hoarseness is a common complaint in todays high strained life[13] India & other developing countries with lower economic status, poor nutrition, poor general health, different food habits, vocal habits, smoking & drinking habits unhealthy environment and different social customs influence the incidence of hoarseness [12] As part of tremendous advancement in field of laryngology. (Microlaryngoscopy & endolaryngeal microsurgery) have already induced considerable rethinking on earlier concepts of certain laryngeal conditions such as those associated with epithelial hyperplasia[12]

AIMS & OBJECTIVES

1. Analyse clinical profile of hoarseness
2. To find out common etiology of hoarseness of voice
3. To find out association of common Predisposing factors leading to hoarseness.
4. To study management of hoarseness of voice.

REVIEW OF LITERATURE

Historical Review

In Corpus Hippocraticum, Hippocrates in fifth century BC speculated that lungs and trachea played a role in the production of voice and that articulation was function of tongue and lips.[14] Claudius Galen (131 to 201 AD) was first to describe the larynx with its three major cartilages and paired muscle, and identified it as the instrument of voice terming it the "principallissimum organum voices".[14] The myo-elastic theory of phonation was enunciated in 1839 by Johannes Mueller who proved that it's the air-stream passing through the vocal folds which are set in vibration and produce sound.[14]

Hermann Von. Helmholtz (1921-1894) pioneered the acoustics of sounds by using very simple experimental models, which have formed the basis of further research in phoniatrics.[14] There was no trace of laryngoscope before middle of eighteenth centuary.[15] The science of laryngology of the present day really took off after the Spaniard singing teacher Manual Garcia (1905-1906) in Paris, first saw his own larynx with aid of dental mirror which cost him only six francs utilizing sunlight.[14] Gracia was awarded father of laryngology for his work on laryngoscope.[16] In the year 1804 AD, Dr. Bozzini of Frankfort created a great sensation throughout Germany with his invention for illuminating the various canals of the body. In 1807 he published a work on the subject.[15]

In1827 Dr. Senna of Geneva invented a little mirror to see the glottis. But he gave up its use on account of the too small size of the instrument.[15] In 1829 Dr. Benjamin Guy of Babingtone exhibited at the Hunterian society of

London an instrument closely resembling the laryngoscope now in use.[15] In 1838 M. Baumer exhibited at the world society of the Larynx, a mirror about the size of a two france piece which he described as being very useful for examining the posterior naris and larynx.[15]

In 1854, the idea of employing mirror for the internal examination of the larynx occurred to Signor Manuel Garcia. The effort of Signor Garcia who was quite unaware that any similar attempts had previously been made in the same direction, were crowned with success, and the following year he presented a paper to the Royal Society of London entitled, "Physiological observation of human voice". Signor Garcia's laryngoscopic investigations were all made on himself. Indeed he was first person who conceived the idea of an autoscopic examination of the larynx. His method consisted introducing a little mirror fixed to a long stem, suitably bent, to the top of the pharynx. He directed that the patient being experimented upon would turn towards the sun so that the iluminious rays falling on the little mirror should be reflected into larynx but he added in a footnote, the observer experiments on himself, he ought, by means of a second mirror, to receive the rays of sun and directed them on the mirror which is placed against the pharynx. In 1857, Prof. Turck of Vienna endeavored to employ the laryngeal mirror in the words of general hospital.[15]

In 1857, Prof. Cezermark of Perth, commenced to work with one of Dr.Turck's laryngeal mirror and in short time overcome all difficulties. Artificial light was substituted for the uncertain rays of sun, the large ophthalmic mirror was used for concentrating the luminous rays and by making them into different sizes. Thus it was Garcia's reinvention of the laryngeal mirror led to Cezermark

to create the art of laryngoscopy.[15]

Morell Mackenzie (1865) of London, a skillful laryngologist and teacher, redesigned the laryngeal mirror and popularized indirect laryngoscope.[17] Gustav Killian, in Berlin developed a direct laryngoscope and an apparatus for suspending the laryngoscope. Later, Sir Felix Seman, Sir St Clair Thomson, V.E. Nagus and Jackson developed the subject of laryngoscope. Killian and Jackson – combined endotracheal anesthesia with laryngoscope and introduced under direct vision and added distal illumination.[18]

In 1953, the Zeiss operating microscope was introduced. Kleinsauser (1961) revolutioned diagnosis and treatment of a laryngeal lesion using microlaryngoscopy. He adapted the binocular Zeiss Microscope to direct laryngoscope, using a 400mm objective lens.[17] Use of stroboscope was first described in1878 by Oertlet. Killian in 1932 described the surgical and optical properties of stroboscopic light .In 1961-Van Laden described use of electronic stroboscope.[19]

In 1968-flexible fibrescopic laryngoscopy was introduced by Sawashima and Hirose and one of the other significant advance in laryngeal examination has been the development of rod lens telescope by Hopkins and its application to laryngeal documentation by Ward et al. and others.[20]

In the early 1970s Jako Strong, Vaughan described coupling of CO_2 laser to surgical microscope and this provided greater precision and facility for endolaryngeal surgery.[21]

In 1960, Theodore made first laser, in 1961-Nd-glass laser, in 1964 Nd-Yag Laser were developed. In 1965 CO_2 laser – was developed.[22] In 1968 Polanyi along with Jako – used the articulated arm and the CO_2 laser to ablate vocal fold papillomatosis. It was from this point that lasers were used in otolaryngology and head and neck surgery and the use has continued to spread, becoming more diverse but yet more specialized in each application.[22]

ANATOMY OF LARYNX

Anatomy of Larynx (Vocal folds)

The vocal folds are defined as two fold like structures, which extends from middle of angle of thyroid cartilage to the vocal processes of arytenoid cartilage. Vocal fold is made up of mucosa and muscle. Mucosa is subdivided into epithelium, which is of stratified squamous type and lamina propria, which consists of superficial, intermediate and deep layers. The superficial layer of lamina propria, referred to as Reinke's space, consists of loose fibrous substance. It is this layer, which vibrates most significantly during phonation. The intermediate layer and deep layer form vocal ligament, deep to which is the vocalis muscle which constitute the main body of vocal fold. Anterior and posterior macula flava, which are mass of elastic fibers, serves as cushions to protect the ends from mechanical damage. [23]

The rima glottides or glottis is an elongated fissure between the vocal folds anteriorly and vocal process and bases of arytenoid cartilages posteriorly. The region between vocal folds, is termed intermembranous part and remainder that lies between vocal processes is called inter cartilaginous part. The glottis alters the shape with phonation and respiration. The average length of glottis in men 23mm and 15-17mm in women.[24]

The muscles

The muscles of the larynx may be divided into extrinsic, which attach larynx to neighbouring structures and maintain the position of larynx in the neck and intrinsic, which move the various cartilages of the larynx and regulate

the mechanical properties of the vocal folds.[23]

Extrinsic muscles

The extrinsic muscles may be divided into those below hyoid bone (infra hyoid) and those above hyoid bone (suprahyoid). Infrahyoid muscles include thyrohyoid, sternohyoid, sternothyroid and omohyoid. Suprahyoid muscles include–mylohyoid, geniohyoid, stylohoid, stylopharynges, palatopharynges and salphigopharynges.[23]

Intrinsic muscles

The intrinsic muscles are of great importance in regulating the mechanical properties of vocal folds as they control not only the position and shape, but also elasticity and viscosity of each layer of the vocal folds.[23] By contraction entire vocal fold is adducted, lowered, elongated and thinned.[23]Posterior cricoarytenoid**,** which is the only muscle to open the (abduct) glottis, arises from lower and medial surface of the back of the cricoid's lamina and fans out to be inserted into back of the muscular process of arytenoid cartilage.[23]

Interarytenoid muscles comprise the unpaired transverse arytenoid muscle and paired oblique arytenoid muscle. Transverse arytenoid muscle arises from the posterior surface of muscular process and outer edge of one arytenoid and passes to similar attachments on the other cartilage. The oblique arytenoid passes from posterior aspect of the muscular process of one arytenoid cartilage to the apex of the other thus crossing each other. Interarytenoid muscle adducts the vocal cord chiefly at cartilaginous portion.[23]

The thyroarytenoid (vocalis) The lower part of muscle is thicker and forms a distinct bundle called vocalis muscle, contraction of which adducts the vocal folds, especially membranous portion.[23] Cricothyroid muscle is the only intrinsic muscle, which lies outside the cartilaginous framework. The action of cricothyroid muscle is to lengthen the vocal folds and vocal folds are brought into paramedian position.[23]

Lymphatic drainage

The vocal folds are firmly bound to the underlying vocal ligaments and this results in absence lymph vessels, a fact which accounts for the clearly defined watershed between upper and lower zones.[23]

Nerve supply

The nerve supply is from vagus by its superior and recurrent laryngeal branches. Superior laryngeal nerve arises from inferior ganglion of vagus and receives a branch from superior cervical sympathetic ganglion. It divides into a small external branch, supplying cricothyroid muscle, and large internal branch divides into two main sensory and secretomotor branches. The upper branch supplies mucous membrane of lower part of pharynx, epiglottis, vallecula and vestibule of larynx. The lower branch descends in the medial wall of pyriform fossa beneath the mucous membrane and supplies aryepiglottic folds and mucous membrane to the level of vocal folds. The internal branch ends by piercing the inferior constrictor muscle of pharynx, unites with an ascending branch of recurrent laryngeal nerve. This branch is called Galen's anastomosis or loop and is purely sensory. [23]

The recurrent laryngeal nerve on the right side loops under subclavian artery and left side, loops around arch of aorta, and ascends upwards accompanied by laryngeal artery enters the larynx. The nerve then divides into motor and sensory branches. Motor branch supplies all the intrinsic muscles except cricothyroid. The sensory branch supplies the laryngeal mucosa below the level of vocal folds and also carries afferent fibers from stretch receptors in larynx.[23]

PHYSIOLOGY OF VOICE PRODUCTION

The human voice is produced by passive vibrations of the vocal folds in an air stream, usually on exhalation. The vibrations produce pressure waves that are received by the usually on exhalation. The vibrations produce pressure waves that are received by the auditory system and perceived as sound, much like any sound source producing pressure waves that are perceived as the sound of a siren, a French horn, or a cello, Normal human voice production requires several elements.

1. An adequate air stream to initiate and sustain vocal fold vibrations.
2. Vocal fold edges that are free from aberrations
3. Vocal folds with normal tissue properties to allow vibrations
4. Optimal posturing of the 2 vocal folds in contact or near contact.
5. Proper internal tension of the vocal folds to produce the desired pitch and voice quality.

If normal voice is a series of pressure waves with a certain shape produced by normal vocal folds, than an aberration on the vibrating edge of a vocal fold, a change in the distance between the 2 vibrating edges, or a change in the internal tension of the vocal folds can all change the shape of the pressure waves, which are than perceived as an altered sound or hoarseness. Each of these elements is addressed in turn.

Prime reason for the existence of the larynx is not to make phonation possible, but to provide a protective sphincter at the inlet of air passages.[23] The human larynx is a highly modified portion of airway. Voice can be produced in this tubular organ by the vocal cords vibrating in an expiratory blast of air. The

vibration of cords effectively chops the air stream into a series of rhythmical segments of puffs. This produces a complex motion of the air column consisting of fundamental tone and overtones. The complex of sound frequencies are modified by the resonating and vocal cavities to impart its characteristic quality or wave from changes in the shape of the resonating vocal cavities. This can transform the laryngeal sound into various vowels and consonant sound, which constitute the vocal components for speech.[25]

Voice Production[25]

Production of voice by the larynx requires three mechanisms:

- The respiratory bellows, which produce the expiratory blast of high-pressure column of air.
- Vibrating mechanism in the larynx.
- Resonating chambers in the thorax and pharynx, mouth and nasal chambers.

The respiratory bellows[25]

The high-pressure subglottic column of air is essential of phonation. This is provided by the contraction of muscles of expiration in the thorax and abdominal wall. Immediately before phonation the cords are adducted and tensed. The muscles of expiration contract and compress the thorax with a rise in thoracic and infraglottic pressures.

When the pressure reaches an adequate level the cords are set vibrating with production of laryngeal sound. The force of magnitude of the air pressure generated in the subglottic space determines the intensity or volume of sound produced. The loudness of the sound is almost directly proportional to the

force of blast of air.

The vibrating mechanism[25]

The vibrating structures essential for phonation are the true vocal cords. In larynx the pitch of the note produced depends on the frequency of vibration of cords. This is regulated by number of factors.

- Regulation of tension of vocal cords.
- Variation in the length of the segment of the vocal folds that is actually vibrating
- Adjustment of the shape of the free margin of the vocal folds. These may vary from broad and thick to thin and narrow. Thus, alteration is accomplished by the action of the internal fibers of the thyroartenoid muscles.
- The pressure of the infraglottic column of air being forced through the vibrating cords. The pitch of the laryngeal tone rises with increasing force of the blast of the air if the cord tension remains constant.

Resonating Mechanism[25]

The tone produced in the larynx is weak and non resonant. Modification and enhancement of the overtones in the laryngeal tone is produced by the resonating mechanisms. This gives the voice its characteristic richness and fullness of quality. The resonators are the air space of the lungs, trachea and the supralaryngeal resonators, the pharynx, the oral cavity, the nasal chambers and the sinuses. The resonators increase the volume of the feeble laryngeal sound, re- enforce some of its overtones and then give the voice its individual quality.

The resonators are not static but dynamic; modification in the shape of these resonators of the pharynx and mouth by the lips, tongue and palate is a mechanism in the articulation of vowels and consonant sounds that constitute speech.

Speech may be summarized as the production of sound by the larynx (phonation) and the modification of this sound by resonance of the supralayngeal air spaces (articulation).

Vocal Cords during Phonation[25]

The production of laryngeal tone is associated with vibration of the cords. The cords are first approximated and rendered tense by contraction of the intrinsic and extrinsic musculature of larynx.

The adducted tense vocal cards are set into vibration as air column passes through glottis. During vibration the cord margins are rolled upwards and outwards. The movement suggests that the surfaces of contact of their margins are being forced apart from below. The lower parts of opposed margins are separated before the upper. The cord movement has a vertical as well as a horizontal component, the cord edges moving on an elliptical path with its long axis horizontal and its short axis vertical.

These vibrations may involve the entire length of cords or segments of their anterior ends, varying with the pitch of the laryngeal tone. The time relations of the opening and closing phases of the cycle of vibration vary with the pitch of the sound. The lower the pitch, the longer the period of closure. With increase in the pitch the period of closure becomes progressively shorter than the duration of the opening phase.

The effect of this vibratory cycle, during which the cords make contact and then separate, in that column of expired air is cut up into a series of rhythmic short columns of air. The vibrating cycle produces, the rapid series of phases of compression and rarefaction of air, which constitutes sound. The frequency of these phases of alteration or pressure changes, determines the pitch of tone. The force or the power of air stream determines the volume of intensity of sound.

Mechanism of pitch variation[25]

Alteration in pitch of the laryngeal tone is achieved by regulation of the length and tension of vibrating segments of the cord, the shape and size of the contact areas of the cord edges and the air pressure.

Length and Tension of Vocal Cords[25]

The shorter the vibrating segment and greater the tension, the higher is the frequency. The length and tension changes in the vocal cords are controlled by the thyroarytenoid muscles and indirectly by extended laryngeal muscle especially cricothyroid which is involved in the production of high-pitched tones. Shape and tension of the vocal folds is adjusted to the pitch of sound before the sound is actually produced.

Shape of the vocal cords[25]

In production of low tones, the vocal cords are broad and vibrate as a whole along their entire length and their tension is relatively low. In the upper range of the vocal scale the edges of the cords are thin and the areas in contact are reduced. During normal conversational speech only shape of the cords and changes in the tension takes place, whereas in the singing cords also lengthen as the pitch raises. This is brought about by the cricothyroid muscles.

Subglottic pressure[25]

The force of the expiratory blast of air, as well as determining the volume of sound, also influences slightly the pitch. Increase in air pressure in associated with increase in pitch, when it is increased in volume. It has been suggested that in the production of very high notes the false cords come into contact with the upper surface of vocal cords and this raise the frequency of their vibrations.

Theories of mechanism of vocal fold vibration

The vocal fold vibration is essential to voice production was demonstrated in the first canine vocal fold experiment performed by Ferrein in 1974. Subsequently, there have been a number of hypothesis about how vocal fold vibration is controlled.[26] Neurochronaxic theory, which attributed vocal fold vibration to an active pulsating muscle contraction of the vocal folds, has been rejected. The currently prevailing view, is the myoelastic-aerodynamic theory of voice production.[26]

Neuromuscular or Clonic Theory[25]

This theory postulates that the vibrations of the vocal are a direct result of active muscle contractions. The vibrations are brought about by a rapid series of separate active contractions of the thyroarytenoid muscle. The vibrations of the cords are not dependent on the air stream through larynx. It is claimed that the thyroarytenoid muscles contains special transverse fibers. The contraction of these transverse fibers separates the approximated cords, while their relaxations allows the cord to recoil by virtue of their inherent elasticity and to close the glottis. Thus

the cord vibrations are maintained by rhythmical cycles of active contraction and relaxation of thyroarytenoid muscles. These rhythmic contractions are the result of excitation by rhythmic discharge of impulse of motor neurons, which in their form are stimulated during phonation by rhythmic bursts of impulse generated in cerebral cortex. All these are at the same specific frequency (thus clonic contraction produces vibrations of the cords at this particular frequency). The end result of this is that the series of puffs of air emerging from the larynx are also at this self same frequency resulting therefore in the production of a tone or intended specific frequency or pitch. The laryngeal air pressure has no modifying effect on the frequency of the movements of vocal cords but it may have a supplementary action in varying the amplitude of these cord movements.

Myoelastic – Aerodynamic Theory[26]

In 1985, Van den Berg proposed two basis principles of the myoelastic – aerodynamic theory of voice production. First, he suggested that the fundamental frequency of vocal fold vibration is determined by a number of interdependent factors, including the mass and viscoelasticity of the vocal folds and the subglottal pressure. Secondly, he proposed that during phonation the vocal folds are driven into vibration by forces that are explained by Bernoulli's principle.

Myoelastic[26]

The myoelastic aspect of the phonatory control refers primarily to the neuromuscular control of vocal fold tension and elasticity during phonation. According to the myoelastic – aerodynamic theory, vocal folds are adducted, contracted, and tensed during phonation to regulate vocal fold elasticity. The coordination of subglottal pressure and vocal fold elasticity is thought to be key to

regulating phonatory output. In addition the neuromuscular control of the vocal folds adjusts the configuration of the glottal aperture.

Aerodynamic[26]

The aerodynamic aspects for the myoelastic – aerodynamic theory emphasizes the role of fluid dynamics in setting vocal folds into vibration once they are adducted. The three aerodynamic principles that are critical for vocal fold vibration are that (1). Air flows from a high-pressure region to a low-pressure region; (2). The pressure of an incompressible flow decreases as the particle velocity of the fluid increases, in accordance with the principle of conservation of fluid energy (Bernoulli's energy law); and (3). The particle velocity of an incompressible flow confined in a duct increases as the cross – sectional area of the duct decreases.

Once air flows through the open glottic aperture, there are three main closure forces. (1). The Bernoulli's effects of airflow through the glottis generate negative force that pulls the vocal fold medially. (2). The elasticity (mainly passive recoiling) of the vocal fold, and (3). Drop in subglottal pressure. These factors together cause the vocal folds to close toward their approximated position. When the closure of the aperture obstructs airflow, a new rise of subglottal pressure builds up until it is again sufficient to deform the vocal fold tissues, and another cycle of the opening phase starts. This cycle of vibration is called the glottal cycle.

DISORDERS OF VOICE

The voice is the primary means of communication for humans both socially and in the work place.[5] Both professional and nonprofessional, visit the otolaryngologists first when aphonia, hoarseness or some other voice or speech problem occurs.[6]

Communication disorders can be grouped into three categories; disorders of voice, disorders of speech and disorders of language.[6]

Causes of voice disorders

Something must be abnormal or atypical in the way in which vocal folds function to produce voice. In such an organ, two vocal folds would have – same dimensions, they would move symmetrically and regularly and each vibratory cycle would include three phases: glottal opening, glottal closure and closed glottis. The vocal sound from this ideal larynx would be judged "excellent", it would be smooth and free from all hoarseness, have an appropriate pitch range for the age and gender of talker, and would be capable of wide pitch and loudness variation.

Abnormal vocal fold vibration may take many forms, stroboscopy have revealed that one fold may move faster than other, vibrations may be limited to one vocal fold, there may be no glottal closure; the vibratory pattern may be dissimilar at different regions along one or both folds and so an. Acoustic indicator of such patterns may include pitch period perturbation, amplitude perturbation, decreased signal to noise ratio, altered fundamental frequency and

many others. The potential complexity of vibratory pattern resulting from combination of these cyclic abnormalities and sequential irregularities is almost endless. Accordingly if hoarseness in its various forms can be pressured to result from abnormal vocal fold vibration, its origin should be found in one or more of the deviations.[6]

Classification of voice disorders

A number of classification proposal have been advanced, but these have been limited to specific types of voice disorders such as muscle tension dysphasia. Traditional texts have also tried to classify voice disorders as functional or organic, or as disorders resulting from vocal fold closure or lack of vocal fold closure. These descriptive categories lack reference to pathologic conditions or the presence or absence of a lesion. No such global classification system exists at present.[27]

Clark A Rosen (2000) Proposed classification & nomenclature and divided voice disorder into 4 Major categories[27].

1. Nonorganic voice disorder (functional)

Nonorganic voice disorders (functional) have a common finding of dysphonia associated with normal vocal fold morphology and normal vocal fold motion. It includes, Muscle tension dysphonia, Conversion dysphonia, Psychogenic dysphonia, Functional dysphonia.[27]

2. Organic voice disorders

Organic voice disorders involve actual pathological changes to larynx in general

and to vocal fold in specific and includes – vocal nodules, polyps, cysts, Reinke' s edema, granuloma, leukoplakia, carcinoma of vocal fold etc.[27]

3. Movement disorder

Laryngeal movement disorders involve abnormal movement of larynx and caused by abnormalities in muscle control. Common disorders within this category are unilateral vocal fold paralysis, spasmodic dysphonia etc.[27]

4. Systemic disease that affect the voice production system

Often systemic diseases have adverse effects on the function of the vocal production tracts and results in a voice change e.g. Reflux laryngitis, infections of larynx, neurological diseases like Parkinson's disease.[27]

Another classification mentioned by P.H. Donste divides voice disorder into two groups.[2]

1. Functional voice disorders
2. Organic voice disorders.

Functional voice disorder again subclassified as[2]

1. Psychogenic (Phononeurosis) disorder – voice is inhibitedby psychological stress
2. Habitual disorder. Faulty use or overloading of voice. There is no laryngeal disease; the phonatory system is capable of function. The cause of dysfunction is either emotional or habitual.

Organic voice disorder[2]

1. Primary organic voice disorder – e.g. congenital web, neuromuscular disorder papilloma, cysts, polyps, trauma, malignances etc.

2. Secondary organic disorders – they are the consequences of temporary chronic abuse of vocal cords and, as long as they have not progressed too far, are still reversible.

Classification proposed by – Fred D. Minifies – based on the characters of voice which may be either functional or organic: Pitch disorder, Loudness disorders, Voice quality disorders and those with mixed symptoms.[6]

I. Pitch disorders[6]

Pitch disorders are present when the voice is consistently higher or lower than would be expected for a particular individual of a given gender and age.

A. Functional pitch problems[6]

Example is the continued use of a high pitched (falsetto) voice by a post-pubertal male. Low pitched ventricular phonation where talker produces a low pitched, gravely voice through vibration of false vocal folds.

B. Organic high pitch problems[6]

Causes are

1. Under developed larynx. Larynx has small vocal folds and vibrate more rapidly and create high pitch. It is seen in hormonal imbalance, hereditary familial body structure, genetic syndromal forms of dwarfism.
2. Laryngeal web. It may be congenital or cicatrical. Its effect is to shorten the free portions of the folds and thereby produce higher pitch.
3. Structural asymmetry. Structural asymmetry may cause the vocal process of one arytenoids cartilage to slide on top of or below, its opposition member such that posterior parts of the membranous folds are pressed together, there by effectively shortening their vibrating portions.
4. Swelling at the anterior commissure. Enlargement of one or both vocal folds adjacent to anterior commissure, shortens the vibrating length of folds and produce higher pitch.

C. Organic low pitch problems.

Most common organic origin of low-pitched voice are Reinke's edema, virilization, glottalization and vocal fry and tremulousness.

3. Loudness disorders[6]

A. Functional

Personal adjustment. Atypical loudness is often indicator of such personality types as the overly aggressive, the shy and socially insure.

Environmental stress. Some people are required to speak loudly in their occupations. This vocal requirement causes laryngeal trauma, subsequently change in vocal organs, and consequent voice disorders.

B. Organic.

Paralysis or paresis, bowed vocal folds, sulcus vocalis, hearing impairment causes organic loudness problems.

4. Voice quality disorders[6]

Voice quality disorders are the most common and complex vocal problems. They encompass resonance and phonatory components, which may be mixed in various ways. Phonatory disorders are presented under following headings. Aphonia, Breathiness, Harshness, Hoarseness, Spasmodic dysphonia

Aphonia, the absence of phonated sound, is revealed as a whispered voice, which indicates that the vocal folds are not vibrating. Aphonia is often is functional disorder. Aphonia can also result from organic diseases.

Breathiness. It is excessively audible breath flow noise that is accompanied by a relatively low vocal loudness level. Vocal cords vibrate during the production of a breathy voice, but do not impede the airflow sufficiently to allow much

increase in subglottal pressure.

Harshness. When vocal folds remain in contact for a disproportionately long in vibratory cycle, a voice quality knows as harshness results. It may be either functional or organic.

Hoarseness. Any condition that alters the regular, repetitive, synchronous vibration of vocal fold causing randomly timed or randomly intense pressure pulses create the voice quality called hoarseness. Physical conditions that cause random aperiodicity include disease or condition in the larynx that changes the size, stiffness, or surface characteristics of one or both vocal folds or that causes excessive squeezing of one fold against the other. Any other these factors may create the coditions for hoarseness.

Spasmodic dysphonia (Spastic dysphonia) The problem originates in the larynx and it is heard most frequently as a sudden momentary interruption of the voice caused by brief, spasmodic glottal closure. In some patients, instead of closing, the glottis spasmodically, opens to allow the air to escape as a whisper it is probably an organic movement disorder of unknown cause. The closure form of the disorder in often referred to as adductor spasmodic dysphonia and the open form as abductor spasmodic dysphonia.

Resonance and Resonance disorder[6]

When shape and adjustments of the resonance spaces do not conform to the customary configuration, resonance disorders are apt to be present. The two most common resonance defects are too much nasal resonance (hyper nasality) & insufficient nasal resonance (hypo nasality).

EVALUATION OF PATIENT WITH HOARSENS OF VOICE

Evaluation of Patient with hoarsens of voice

Evaluation of patient with hoarseness of voice or any dysphonia is multi disciplinary approach, which involves – otolaryngologists, speech pathologist, speech therapist, neurologist etc, which provide proper diagnosis and management,[20] where as voice laboratory provide functional diagnosis.[28]

1. History of Complaint

When taking the history from patient, principle complaint is elicited first, in patient's own word. It is supplemented on the following points like date of onset, gradual or abrupt, the course, and previous treatment. What was the voice like before the trouble begin; earlier similar troubles; which activities in patient's job or free time put demands as voice? For comparison at later visits the qualities of voice should be recorded in writing. Is the voice: Low, Loud, Powerful, Clear, Sharp, Resonant Periodic, Relaxed, or High, Soft, Weak, Breathy, Hoarse, Dull, Thin, Falsetto, Raw, Harsh, Tense, Strained.[2]

It is important that patient attempt to recall what may have led to current vocal problem.Upper respiratory tract infections are commonly linked to the onset of voice disturbance. Also a viral illness or upper respiratory tract infection may precede vocal fold paralysis. For example a herpetic infection may cause recurrent laryngeal nerve neuropathy, resulting in paralysis.[29]

A history of trauma preceding the development of dysphonia is usually obvious. These patients often note hoarseness several weeks following trauma.[29]

Medical history. The laryngologist must relate respiratory or other illness that may have proceeded or may be associated with vocal problem, including

asthma, emphysema and chronic bronchitis.Because the lungs are the power source for voices, reduced maximum phonation time and early vocal fatigue.[29,30] Endocrine disorders like hypothyroidism, diabetes; stress related disorder; puberty, menopause, menstruation (laryngiopathies premenstrualis) cycle change; Autoimmune disorders; Neurological disorder; gastro intestinal disorders. Gastro esophageal reflux disease is now generally accepted as a significant factor in a number of pathologic laryngeal processes.[29]

Past surgical history, including vocal fold surgery, also include any portion of body particularly abdominal, thoracic surgery; neck surgery.[30] Endotracheal intubation, during general anesthesia should be enquired.[29]

Social history, smoking, ingestion of alcohol, high consumption of products containing caffeine, fat, spicy food which contribute laryngophryngeal reflux etc enquired.[30]

Occupational history, Excessive voice use in occupation, working in loud environment, exposure to chemicals in the workplace can contribute to the disorder. Increased vocal demand results in dysphonia.[30]

Vocal abuse. With the patient exhibiting poor or detrimental vocal habits.[30] Some patient may report that their voice disorder developed after a traumatic life event, such as death of loved one.[19]

Physical examination

The physical examination of a patient with vocal complaints must include a complete car, nose and throat examination to ensure that three is no gross pathological condition. Also a gross check of the cranial nerves and eye should be performed, particularly conjunctiva, to check for chronic irritation[30]

Examination of ear must include assessment of hearing activity. Even a relatively slight hearing loss may result in voice strains[31] .A sensori- neural hearing loss can cause voice problems related to pitch, tone and volume, where as a patient with a conductive hearing loss may produce sounds that are louder than he perceives.[30]

Nose should be assessed for patency, mucosa, secretion, pale gray, allergic mucosa or swollen infected mucosa in nose suggests abnormal mucosa elsewhere in the respiratory tract.[30,31] There may be sinusitis that is part of a chronic, longstanding problem.[31]

Examination of oral cavity should include careful attention to tonsils and lymphoid tissue, mucous. Dental examination should focus on oral hygiene.[32] Neck should be examined for masses, restriction of movement, excessive muscle tension, scar from previous surgery, or trauma.[31,32] Practical attention should be paid to thyroid gland. Cranial nerves should be examined. Diminished fifth nerve sensation, diminishes gag reflex, palatal deviation, or other cranial nerve defects may indicate mild cranial polyneuropathy. Post viral infection neuropathies may involve superior laryngeal nerve and cause weakness, fatigability and loss of range, projection in the voice.

Laryngeal examination

Examination of larynx begins when the patient enters the physicians office. The quality of voice should be noted.[32]

Indirect laryngoscopy (IDL)

Classical mirror examination of larynx remains the preferred technique.[33] This method is known universally and has been used by

otolaryngologists for years. This is typically first procedure used in otolaryngologists office to view the vocal folds. It is quick and requires only mirror and standard lighting.[34]

Advantages

- Allows gross examination of larynx and hypopharynx[35]
- It allows to grossly assertion the motion of vocal cords and balance of vocal fold effort during phonation.[30]
- Provides three-dimensional viewing and good colour resolution.[36]
- Provides better view of posterior portion of vocal cards than is obtained by flexible endoscopy[31]
- Assessment of mucosal surface larynx and hypopharynx, any narrowing,web formation, or mucosal irregularly with in larynx[33], presence or absence of nodules, mass lesion, contact ulcers, hemmorhage and paralysis and other anatomical abnormalities can be established.[35]

Disadvantages

- This method of examination is limited in comparison to newer methods..
- It is difficult to perform – stroboscope and video documentation, for evaluation physician must remember the lesion and document with simple sketch.[36]
- Anatomical limitations are caused by – epiglottis, base of tongue.[36]
- In 5-10% of patient is not possible due to gag reflex.[33,34]

- Subtle lesion go undetected[34]

Trans – nasal flexible laryngoscopy

Utilizing the flexible laryngoscope, which also comes in various makes, it is indeed biggest advancement that is now available for diagnosis and giving insight into voice and its disorders.[13,31,35] It can be performed as office Procedure[34,37] using local anasthesia.

Rigid televideoscopy

- Rigid televideoscopy is done in cooperative patients with local anasthesia to visualize larynx and pharynx. Generally 70^0 or 90^0 Hopkins rod telescopes are used.[14]

Direct laryngoscopy (DLS)

- Rigid endoscopy with anasthesia is reserved for rare patients whose vocal folds cannot be assessed adequately by other means or for patient who needs surgical procedures to remove or biopsy laryngeal lesions.[32]
- It is mandatory in any patient with hoarseness or dysphasia who falls into a high risk group for malignancy, especially smokers, and drinkers over 40 yrs.[33]
- In many cases it may be done with local anesthesia. But usually general anesthesia is preferred.[33]

Micro laryngoscopy (MLS)

Micro laryngoscopy, under general Anaesthesia is mainly utilized for therapeutic reason and facilitates detailed examination of the larynx. A high quality-operating microscope is employed with a 4000mm focal length lens in place. The broad lumen kleinhauser laryngoscope is used for adults while stortz paediatric or Benjamin laryngoscopes give excellent access in children.[33]

Recently new technologies have been developed to enhance the endoscopic information during micro laryngoscopy, eg, rigid angled endoscopies, contact endoscopy, fluorescence endoscopy with or without fluorescence enhancing agents for some cases only, to assess the precursors lesion and their delination.[28]

Stroboscopy

Stroboscopy is based on Talbot's law, which states that an image persists for 0.2 sec on retina after exposure and sequential events lasting milliseconds are not perceptible.[19,35]

Using pulsed light source i.e. strobe light source vocal folds are illuminated. Synchronization of the illumination with the frequently of the vibration i.e. phonation results in an apparent standstill of the vocal cords in any desired position. Similarly slight desynchronization of the frequency of illumination with frequency of vibration will result in an illusion of slow motion of vocal cords. The slow motion effect is created from a montage of several different cycles illuminated at slight different points within cycle.[2,19,33,34]With stroboscopy finer characteristics of the vocal cords during phonation i.e. symmetry; regularity (periodicity) mucosal wave, amplitude of vibration, glottic closure and non-vibrating portions can be accurately studied.[2]

Stroboscopy is best done in conjugation with video recording called video laryngo stroboscopy (VLS) and it gives instant replay and frame by frame analysis. VLS can be done through a rigid endoscope (Hopkins rods) or fibroscope.[2,33,38]

Objective tests

Electroglottography (EGG) Electroglottography or electrolaryngography is a method for monitoring vocal fold contact, rate of vibration, and pertuberation of regularity during voice production.[28,30]

Photoglottography: It is designed to show the changes in the glottal area during phonation. It is a complementary to EGG.[28]

Electromyography: Invasive procedure.[31] Useful in determining inefficiency in neural function of one or both cords.[30] Laryngeal electromyography can determine the prognosis for spontaneous recovery. It helps to localize the specific muscle for the injection of botulinum toxin to treat spasmodic dysphasia.[39]

Videokymography: Videokymography is a laryngeal imaging technique that allows real time evaluation for the vocal fold vibration using a real time scan camera. This camera captures a small segment of the image of vocal fold vibration thus enables to see real time vibratory activity of a small portion of glottis. It may have clinical application for small, subtle irregularities and phase asymmetries in patients with dysphonia that is not entirely obvious after videolaryngo strobosocpy.[34]

Recording voice sample: Recording voice sample has several advantages

.It provides a document for later comparison, acoustic analysis, some features of voice can be discussed with patient to make aware of the nature of dysfunction and motivate him to accept the treatment.[2,28]

Acoustic analysis: It is a non-invasive procedure and reflects the status of vocal function. Acoustic parameters provide objective measurement of vocal function and useful in monitoring changes in voice quality over time; before and after treatment.[28]

Perceptual analysis: Severity of the hoarseness can be quantified using GRABS scale, G-grade, R-Roughness, A-asthenicty, B-breathiness and S-strain on a 0 to 3 scale. The rating is made on current conversional speech or reading a passage. Grade 0 - Normal at absence of deviation, Grade1-Slight deviation, Grade2-Moderate deviation, Grade 3-Sever deviation.[28]

Aerodynamics: Aerodynamics analysis provides information regarding vocal efficiency and normal effort.[28]

Phonetogram: It is examiners subjective assessment of the loudness, pitch and quantity of the voice. Here sound intensity and pitch are plotted against each other in a graph called phonetograph.[2]

Dynamic voice assessment: Dynamic voice assessment is a detailed, thorough voice evaluation protocol using trasnansal flexible endoscopy with a variety phonatory and vegetable activity.[34,37]

AETIOPATHOLOGY OF HOARSENESS OF VOICE

Chronic hoarseness of voice

Chronic laryngitis

Any chronic non-specific inflammatory reaction of the laryngeal mucosa may be called chronic laryngitis.

Aetiology: Chronic laryngitis primarily affect middle-aged men, median age is approximately 57yrs. The exogenous stimuli are physical, chemical or infection and most important being inhaled irritants and notably cigarette smoking. Another factor with smoking is alcohol. Chronic infection of upper or lower respiratory tract is another factor. Vocal abuse in an important physical

factors due mechanical irritation.[40] Allergies, exposures to chemicals in workplace also contribute.[5]

Endogenous factors may be metabolic or constitutional short, heavily built are prone for chronic laryngitis. Diabetic, hypothyroidism and vitamin A deficiency can also contribute.[40]

Reflux laryngitis has gained much attention although the concept is not new chronic inflammation caused by reflux of acid and pepsin, called laryngophryngeal reflux often leads inflammatory changes of the posterior larynx.[5]

Clinical features: Hoarseness is the most frequent and often the only symptoms. Typically the patient complains the voice in worst in the morning.

Chronic laryngitis is diagnosed from history and indirect laryngoscopy.

Granulomatous diseases of larynx

The inflammatory processes that involve the larynx can take many forms, with granulomatous disorders invariably representing the end consequences of chronic diseases.[41] Both specific and nonspecific granulomatous diseases can be found in the larynx.[40]

1.Tuberculosis of larynx[42,43,44,45]

2.Leprosy[40,46]

3. **Syphilis**

4.Scleroma of larynx

5.Amyloidosis[47,48,49,50]

6.Sarcoidosis[51]

7.Wagener's granulomatosis

Fungal infection of larynx

Fungal laryngitis has been often described in immunocompromised patient but is rarely reported in immunocompetent host. Fungal laryngitis usually occurs when the normal host immune response are depressed. Such as during immune suppression or diabetes mellitus, AIDS or when local protective mucosa barrier is impaired as with radiotherapy, inhaled steroids or smoking, trauma.[52]

Most common cause of fungal laryngitis is candida.[51,52]Other responsible organisms Blastomyces, Histoplasma or coccidiodes species, Paracoccidomycosis, cryptococcosis, aspergillosis, rhinosporiodosis.

Definitive diagnosis is made by demonstration of fungal spores, hyphae and pseudohyphae within upper epithelial layer, either by tissue biopsy or culture.

Parasite infections

Parasite diseases in the of larynx are very rare, leishmaniais, trichinosis, schistosomiasis and ascariasis are all parasitic infections which can be in larynx. Lesion of larynx results as granulomatous type.[40]

Edema of Larynx

1.Reinke's edema[54,55]

Etiopathology

The aetiology of Reinke's edema is not completely understood[54]. It is strongly associated with smoking, vocal abuse, laryngopharyngeal reflux, chronic sinusitis, allergy.[36,40,55]

Clinical features

It is commoner in male patients aged between 30 to 60 yrs. Hoarseness is most common symptom, rarely stridor.On indirect laryngoscopy edematous swelling of vocal cords and vocal cords are red, swollen and have a slightly translucent appearance. Sometimes mucosa becomes redundant and polypoid projections are visible[31]

Treatment consists of combination of surgery and vocal rehabilitation.

2.Angioneurotic edema[28,56]

Angioedema occurs with multiple precipitating factors such as temperature extremes, trauma, food, allergy, medications, angiotension converting enzyme inhibitors one of the most common causes.[56] Management consists of discontinuation of inciting agent, antihistamines, and intravenous steroids and in C-1esterase deficiency intravenous C1-iNH is given.[56]

OTHERS

1.Laryngeal granuloma[57]

Treatment: Treatment is simple removal by microlaryngoscopy and recently CO_2 laser in also used.[40]

2.Contact ulcers, conract pachydermia

Treatment: Anti reflux regime, voice therapy, voice rest are primary modality of therapy, microlaryngoscopic excision is last resort.

3.Laryngocele[58,59,60]

Treatment: The best operation for laryngocele aims at excising the saccules at its neck. Internal laryngocele are uncapped, if recurrence occurs they should be excised with approach used for external laryngocele.[59]

4.Vocal nodules[61,62,63]

They occur most commonly among male children and female adults. They arise at the junction of anterior and middle third of vocal folds. They result in hourglass closure of glottal configuration and will affect vocal fold mucosal wave and vibration.[62]

Aetiopathology: Vocal nodules are superficial lesion of the lamina propria and pathological condition is thought to occur at basement membrane and superficial layer of lamina propria.[63]

Clinical features: Patient presents with either chronic hoarseness or repeated episodes of acute hoarseness.

Treatment: First line of treatment is behaviour (voice) therapy. Surgical removal, when nodules persist and voice remains unacceptably impaired from patients perspective, after adequate trail of voice therapy.

5.Vocal folds cyst[62]

Aetiology is vocal abuse, misuse or overuse[62] and patients normally presents with hoarseness of voice.[36]

Treatment is unroofing the cyst or marsupilization.[36] Best treated with epithelial cordotomy and microflap approach to preserve as much as vocal fold to optimise postoperative vibratory mechanics.[62]

6.Vocal fold polyps

Vocal cord polyps are more commonly unilateral, translucent, red, pedunculated arise in free edge of anterior third of vocal fold.[62]

Aetiopathology

They are due to vocal abuse, overuse, misuse.[62]

Treatment includes voice therapy, surgical removal[36,61] and low dose, steroids[31]

7.Autoimmune disorder

8.Ventricular phonation

It may occur as a manifestation of hyperfunction with extreme constriction of the entire vocal tract or as compensation for true vocal fold dysfunction. Usually voice therapy is given.[64]

9.Psychiatric conditions

10.Presbylaryngeus

Presbylaryngeus is most common cause of hoarseness of voice in elderly. Women are more affected by these changes; estrogen administration in postmenopausal patient may delay the onset of presbylaryngeus.[5]

11.Arthritis of cricoarytenoid joint[65]

Arthritis involving cricoarytenoid joints may impair the motion of arytenoid cartilages eventually resulting in vocal fold immobility.

12.Radiation therapy

13.Endocrine abnormalities

a)Hypothyroidism and Hyperthyroidism

b)Menstruation

c)Pregnancy

14.Laryngopharyngeal reflux (LPR)

Gastroesophageal reflux (GER) has been implicated in the pathogenesis of

several otolaryngological disorders, Many of symptoms associated with LPR-are easily confused with allergic or sinonasal disease, hence LPR can be overlooked on examination; most common signs will be erythema of arytenoids and interarytenoid area.[66]

Therapeutic approaches include lifestyle modification, acid suppressive therapy and surgical therapy.[66]

15.Laryngeal trauma

There are basically two types of laryngeal trauma penetrating wounds and blunt trauma.[67]

Penetrating or open injures are usually secondary to a knife or gun shot wounds. Knife injuries are generally clean and have sharp edges. Gunshots wound are associated with extensive tissue loss. Treatment requires resuscitation and exploration, meticulous repair.[67] Blunt injuries usually caused by motor vehicle accidents, sport injuries and assaults.[67,68]

Management: Soft tissue radiography of neck, chest, CT scan and endoscopic examinations help to assess the extent of injury.[68]

In cases of blunt laryngeal trauma resulting in no major injuries to cartilage, conservative measures, such as administration of corticosteroids and antibiotics, resting the voice and use of a vaporizer, represents the therapy of choice.[67,68,69] Patient also requires speech therapy in order to reduce the risk of secondarily disturbed vocal compensation and avoid pursuant restriction of phonation.[69]

16.Vocal fold paralysis

Vocal fold paralysis in regarded as a sign of other pathologic finding until

investigations have proven that there is no lesion to explain the paralysis. The left recurrent laryngeal nerve is more frequently involved, because the longer course of the nerve. Incidence of paralysis increase with age, this is likely a result of increased incidence of cancer and neurological damage[70]

Etiology

Etiologies includes surgery, trauma, neoplastic, endotracheal incubation, viral, drug induced and miscellaneous.[72]

Pathology

. Semon's theory, Wagner – Grossman theory (1980) are the postulated pathology.

Presentation: Unilateral vocal paralysis leads to glottic incompetence and hence hoarseness and breathiness of voice.[74] **Diagnosis:** Clinical evaluation includes, history, laryngeal examination, vocal capability assessment, radiographic workup include CT/MRI- brain, skull base to upper mediastinum, panendoscopy.[71]

Surgical methods are vocal fold injection (Teflon), medialization (laryngoplasty), arytenoid rotation, and reinnervation.[71,74]

In bilateral abductor palsy tracheotomy is highly effective mode of management and other procedures are cordectomy, submucoal resection of vocal fold, endoscopic lateralisation, laser assisted arytenoidectomy, reinnervation woodman operation.

17.Tumors of larynx

Tumors of larynx may be benign and malignant.

Benign tumors of larynx

A)Mesodermal tumors Vascular tumors[75]

1.Haemoangioma[76]

2.Chondroma[77,78]

B.Myogenic tumours

a)Leiomyoma

b)Rhabdomyoma

c)Granular Cell tumour[79]

d)Fibroma

e) Lipoma[76]

f))Laryngeal myxoma:[80]

C.Benign ectodermal tumours:

a)Adenoma

b)Paraganglioma[81]

c)Neurogenic tumours

D.Epithelial tumours

a)Laryngeal papilloma

Laryngeal papillomas are most common benign tumours in the larynx,[82,83] and clinically divided into adult onset and juvenile onset forms. Childhood onset recurrent respiratory papillomatsis (RRP) most often diagnosed between 2 and 4yrs of age. Adult onset peaks at 20 to 40 yrs and men are most affected.[82]

Etiopathology: The most common types are HPV-6 and HPV 11. An association between cervical HPB infection in the mothers and incidence of RRP

has been well established. Adult onset papillomas could reflect either activation of virus present since birth or an infection acquired in adolescent life.[83]

Clinical features: The hallmark of papillmatosis in children is triad of relentlessly progressive hoarseness; stridor and respiratory distress. Juvenile type is more aggressive than adult onset. The course of the disease is unpredicatable. Although spontaneous resolution may occur, there is a tendency for recurrence and progression despite all forms of treatment[95]

Treatment: Though surgery is treatment of choice it is not curative. Traditionally removed through microlaryngoscopic techniques.

2.Malignant tumors of larynx

Laryngeal cancer is diagnosed relatively early because it alters the phonatory and airway function.[84] There has been steady rise worldwide incidence, this is attributed to increased tobacco and alcohol consumption.[85]

There is greater predominant in men compared to women 6:1, incidence increase with age, in men between 50-70yrs, for glottic malignancy peak is 62yrs.[75] There is increase incidence in female patients and it may be related to increased use of tobacco and alcohol.[84]

Aetiology: Cause of cancer of larynx is not known,[75] multifactorial theory is proposed due to changing pattern of disease with time and exposure to comparable risk factors.[84]

Gastroesophageal reflux has been proposed as possible co-factor in laryngeal carcinogens.

Histology

The vast majority of malignant tumours arising in the larynx are squamous

cell carcinoma, account for 90% of cases.[84]

Presentation

Hoarseness is almost universal, persistence of this symptom is a important risk factor in early detection.[85] Pain referred to ear is an omnious sign, indicates cartilage invasion. Dysphagia is late feature and indicate advance disease and is feature of supraglottic lesions. Swelling in the neck can occur as a result of either cervical lymphadenopathy or direct extension of disease. Cough, hemoptysis, throat irritation are non-specific early symptoms.[95]

Diagnosis

General ENT examination, endoscopic examination and biopsy for histopathological conformation. CT/MRI assess the size and extent of lesion. Neck examination for lymph node assessment and general examination of chest and abdomen to exclude secondary and second primary[95]

Staging[87]

International union against cancer (UICC) classification is based on anatomical sites of lesion. It's prevent TNM staging is a follows.

Supraglottis

T1 One subsite, normal mobility

T2 Mucosa of more than one adjacent subsite of Supraglottis or glottis or adjacent region outside the Supraglottis; without fixation

T3 Cord fixation or invades postcricoid area, pre-epiglottic tissues, paraglottic space, thyroid cartilage erosion.

T4a through thyroid cartilage; trachea, soft tissues of neck;

deep/extrinsic muscle of tongue, strap muscles, thyroid, oesophagus

T4b Prevertebral space, mediastinal structures, carotid artery.

Glottis

T1 Limited to vocal cord (s), normal mobility a. One cord b. Both cords

T2 Supraglottis, subglottis, impaired cord mobility

T3 Cord fixation, paraglottic space, thyroid cartilage erosion.

T4a Through thyroid cartilage; trachea, soft tissues of neck: deep/extrinsic muscle of tongue, strap muscles, thyroid, oesophagus

T4b Prevertebral space, mediastinal structures, carotid artery.

Subglottis

T1 Limited to suglottis

T2 Extends to vocal cord (s) with normal/impaired mobility

T3 Cord fixation

T4a Through cricoid or thyroid cartilage; trachea, deep/extrinsic muscle or tongue, strap muscles, thyroid, oesophagus

T4b Prevertebral space, mediastinal structures, carotid artery.

All sites

N1 Ipsilateral single ≤ 3 cm

N2(a) Ipsilateral single >3 to 6 cm

(b) Ipsilatral multiple > 3 to 6 cm

(c) Bilateral, contralateral ≤ 6cm

N3 > 6cm

Stage Grouping[87]

Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III T3	T1, T2 No, N1	N1 M0	M0
Stage IVA T4a	T1, T2, T3, No. N1, N2	N2 M0	M0
Stage IVB Any T	T4b N3	Any N M0	M0
Stage IVC	Any T	Any N	M1

Treatment[74]

Over 95% of patients with laryngeal carcinoma are treatable. The causes of untreatability include distant metastesis, poor general health, and refusal by patient, advanced tumors with bilateral node.The management of laryngeal cancer varies in different parts of world, even within one country.

Treatment planning may fall into following categories.

Curative intent:

Surgery

Radiotherapy ± chemotherpy,

Surgery with post operative Radiotherapy or chemo radiation.

Rehabilitation

Palliative

General Palliative care, symptom control and nutritional support

Tracheostomy Palliative surgery Radiotherapy Chemotherpy Chemoradiation

TREATMENT OF HOARSENESS OF VOICE

Medical treatment

A number of mucolytic and mucokinetic like ambroxol, carbocystine and brotuxine are believed to enhance thin the secretions, thereby, supplementing therapy.[4]

Systemic antihistamine decongestant combinations may be required to treat, but otherwise they should be avoided to prevent further dryness. Short-term corticosteroids have been indicted in a number of cases and they facilitate by reducing the oedema of vocal cords there by reducing the hoarseness.[4] Appropriate measures for acid reflux should be taken.

Voice therapy or vocal Rehabilitation.

Voice therapy is a major treatment modality for almost all types of dysphonia. It may be sole treatment of certain voice disorders, or it may proceed and follow pharmacological or surgical intervention.[88] Other key elements include vocal hygiene, medical problems that can affect voice, the importance of smoking cessation, the dangerous and harmful effects of alcohol and drugs, the importance of nutrition and hydration, the effect of voice stress, and useful regular voice exercise program and dangerous of signing sick.[89]

Two general types of instruction relate specifically to direct voice therapy. The first is recovery and second training.[6]

Recovery procedures presume a need for healing, for a return of structures to normal. If the recovery procedures have allowed the larynx to be normal then it is followed by training that modifies previous habit patterns and replaces them with more efficient phonatory behaviours.[6]

Surgery

The role of surgical intervention depends on the cause of hoarseness. Benign mucosal disorders like vocal card nodules, polyp, etc are excised by using microlaryngeal surgery with help of microscopes, laryngoscopes and microinstruments[4] Introduction of phonomicrosurgery using infusion techniques and micro flap technique have revolutionized the voice surgery. The principle is maximum presentation of layered microstructure of vocal fold that is the epithelium and superficial lamina propria. These procedures are designed to improve aerodynamic efficiency and vocal quality by creating a smooth vocal fold edge that is not excavated with overlying flexible epithelium.[90] Laser may be used with great precaution and precision. In vocal fold paralysis, medialization techniques, thyroplasty, provide excellent results.[4]

In case of true benign tumors, removal should be completed but conservative to spare voice, with surgical approach determined primarily by tumour size and location.[36]

Laryngeal cancer and vocal rehabilitates

A. Engineering approach

Engineering approach involves development of artificial sound generator. Breath activated instrument are powered by the breath stream, in which breath from tracheal stoma is carried to a capsule where membrane vibrates and generate sound, on carrying this sound to mouth, it is articulated into speech.[6]

Electrical artificial larynges are battery powered oscillator that generates a focused signals that penetrate the neck and it is articulated in mouth for communication. The most common new generation electronic voice aids are

lighter, smaller more efficient and user friendly. Speech is typically louder, faster intelligible.[91]

B Esophageal speech

The mechanism relies upon the subject charging the oesophagus with air and utilizing vibrations, at cricopharyngeal sphincter level. Any patients who has undergone total laryngectomy have been able to develop functionally satisfactory oesophageal speech. The preoperative and postoperative assistance of a speech therapist is essential in most cases.[6]

C Tracheo-esophageal puncture (TEP)

The wall between trachea and upper esophagus is punctured and a tube of about 4 mm diameters ending in valve is inserted into opening. When tracheal stoma is closed with a finger exhaled air passes through the shunt into esophagus, where it vibrates pharyngo-oesophageal junction. Bloom –Singer, Pange, Provox are replaceable valves, which provide quality phonation. Provox is most widely used.[75]

MATERIALS AND METHODS

The voice is the primary means of communication for humans. Although rarely life threatening, voice problems cause tremendous alterations in daily living and should not be underestimated. A complaint of hoarseness may represent serious disease and therefore should not be ignored, especially if present for more than 2 weeks. It needs a complete evaluation to rule out malignancy as a cause.[5]

Study setting

This Prospective study was carried out in Department of ENT, Tirunelveli Medical College hospital, Tirunelveli.

Study duration

This study was carried during July 2011 to August 2012.

Study design

Prospective study was conducted during 1 year study period.

Study population

Patients attending ENT department of Tirunelveli Medical College hospital, Tirunelveli and also patients referred from other department in the same hospital. A total of 60 cases were studied during the study period.

Inclusion criteria

Patients presenting with hoarseness of voice of ≥ 2 weeks duration.

Exclusion Criteria

1. Patients with hoarseness of voice < 2 weeks of duration.
2. Voice disorders other than hoarseness like rhinolalia aperta, rhinolalia clausa etc.

Methods of data collection

The Proforma was designed based on objective of the study and pretested and used after modification. As per proforma detailed history was taken followed by thorough ENT and systemic examination and clinical diagnosis was arrived with help of relevant investigations.

If findings were inadequate to arrive at a clinical diagnosis, Direct laryngoscopy, videolaryngoscopy with or without biopsy, suspension layngoscopy with or without biopsy was performed. The biopsy specimen obtained was sent for histopathological examination.

Treatment

Treatment was based on the etiology of hoarseness of voice. Medical treatment, including medications like antibiotics, antireflux medications, steam inhalation, voice rest, speech therapy, prescribed for conditions like chronic laryngitis and early case of vocal nodules. Patients with vocal cord polyp papilloma, vocal modules lesion like polyp underwent suspension microlaryngoscopic surgery followed by voice rest and speech therapy. Patients with vocal cord palsy, no specific Rx was given. Patients with malignant vocal cord growth were referred for radiotherapy, as most of them were late presentations. Patients were followed up regularly for three months.

Analysis of data

Data thus obtained was analyzed and presented in the forms of tables, figures, graphs and diagrams wherever necessary.

The findings are discussed in the light of findings in other similar studies conducted elsewhere based on the objective of study in the forgoing chapters.

RESULTS

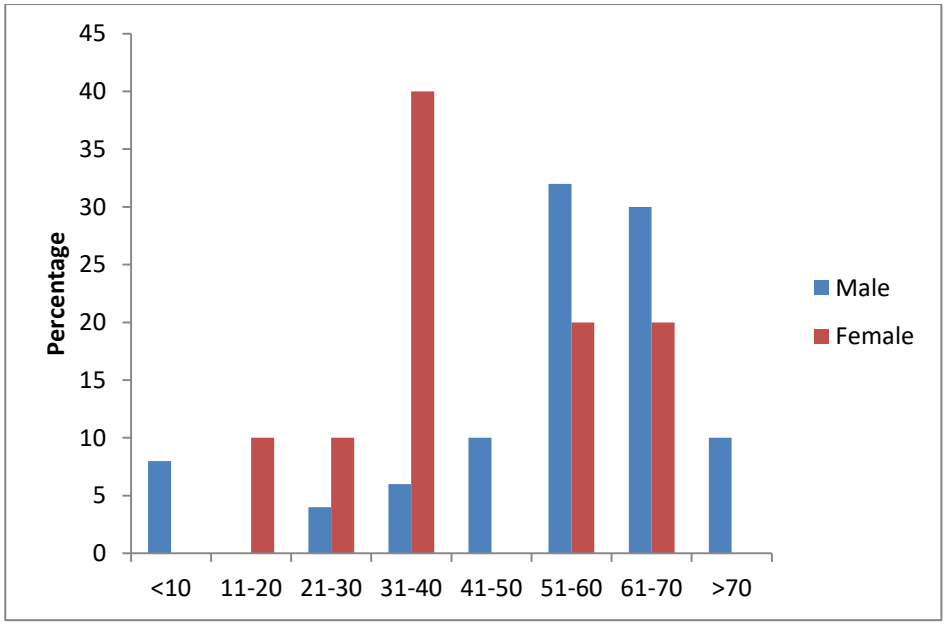
A total of 60 cases were studied during study period.

Incidence

A total of 21,600 cases attended ENT OPD during study period, out of these 60 cases presented with hoarseness of voice. Thus the incidence is 0.27% of all cases.

Table: 1
Age and sex distribution-table1

Age	Male		Female		Total	
	N	%	N	%	N	%
<10	4	8	0	0	4	6.7
11-20	0	0	1	10	1	1.7
21-30	2	4	1	10	3	5
31-40	3	6	4	40	7	11.7
41-50	5	10	0	0	5	8.3
51-60	16	32	2	20	18	30
61-70	15	30	2	20	17	28.3
>70	5	10	0	0	5	8.3
Total	50	100	10	100	60	100



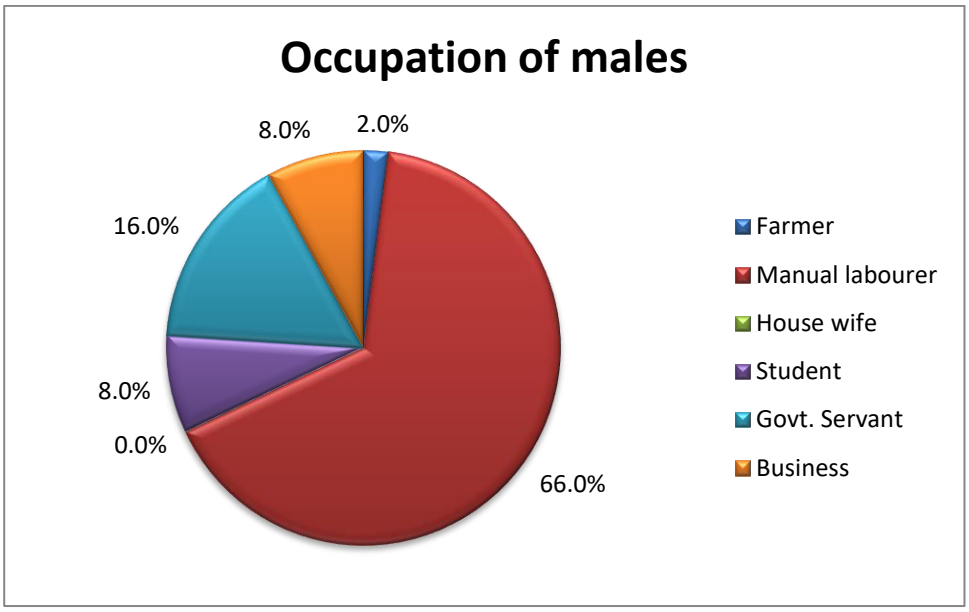
60 cases were studied during one year study period. Maximum number i.e. 18 cases (30%) belonged to 51-60 years of age group and minimum number i.e. 1 case (1.7%) belongs to 11-20 years of age group. 50 cases (83.3%) were males and 10 cases (16.7 %) were females. Youngest patient was of 5years old and eldest patient was of 80 years of age.

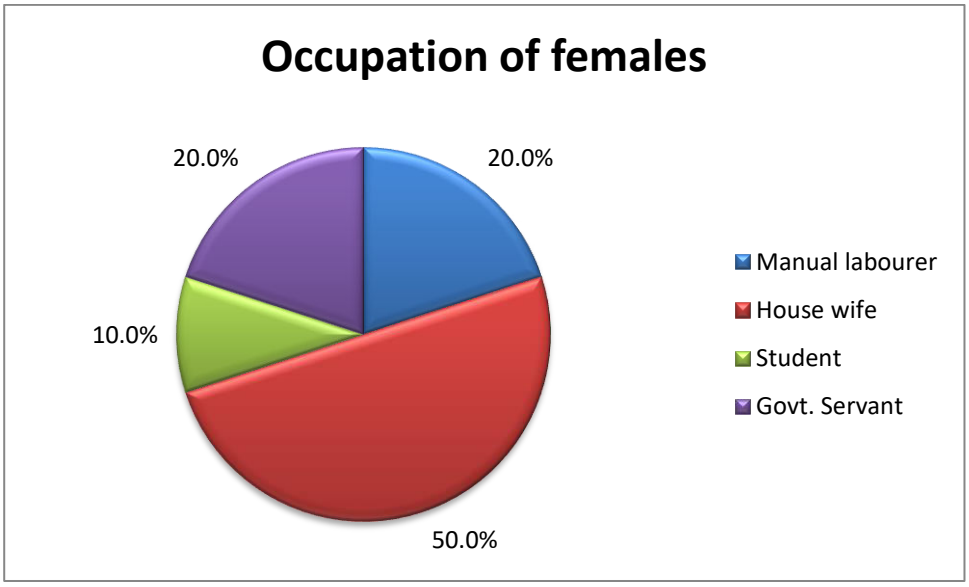
Table - 2

Occupation

Occupation-table2

Occupation	Male		Female		Total	
	N	%	N	%	N	%
Farmer	1	2	0	0	1	1.7
Manual labourer	33	66	2	20	35	58.3
House wife	0	0	5	50	5	8.3
Student	4	8	1	10	5	8.3
Govt. Servant	8	16	2	20	10	16.7
Business	4	8	0	0	4	6.7
Total	50	100	10	100	60	100



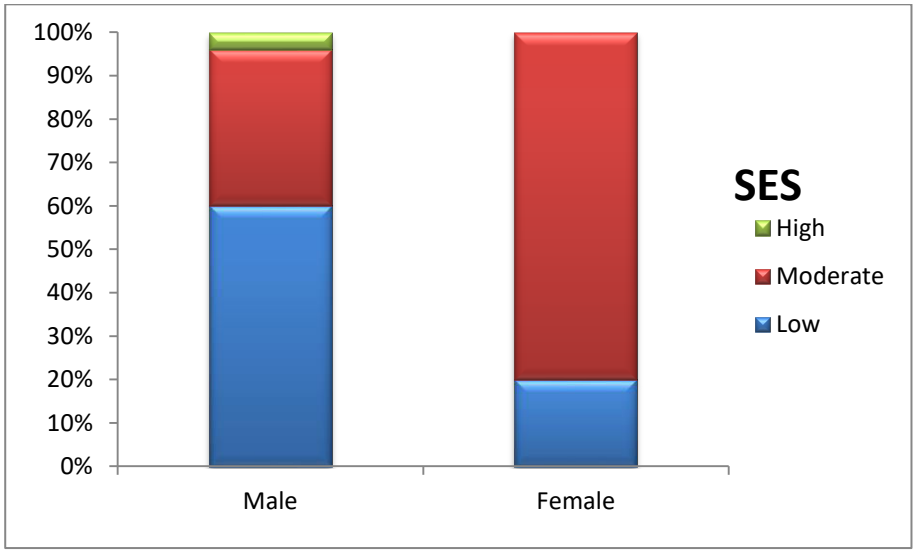


Out of the 60 cases, majority of patients i.e. 35 cases (58.3%) belonged to labour class and the least i.e. 1 cases (1.7%) were in farmers group .Among males labour class predominated i.e. male 33(66.%) and females 2(20%). Among females commonest group house wife 5 (50%).

Table - 3

Socio economic status

SES	Male		Female		Total	
	N	%	N	%	N	%
Low	30	60	2	20	32	53.3
Moderate	18	36	8	80	26	43.3
High	2	4	0	0	2	3.3
Total	50	100	10	100	60	100

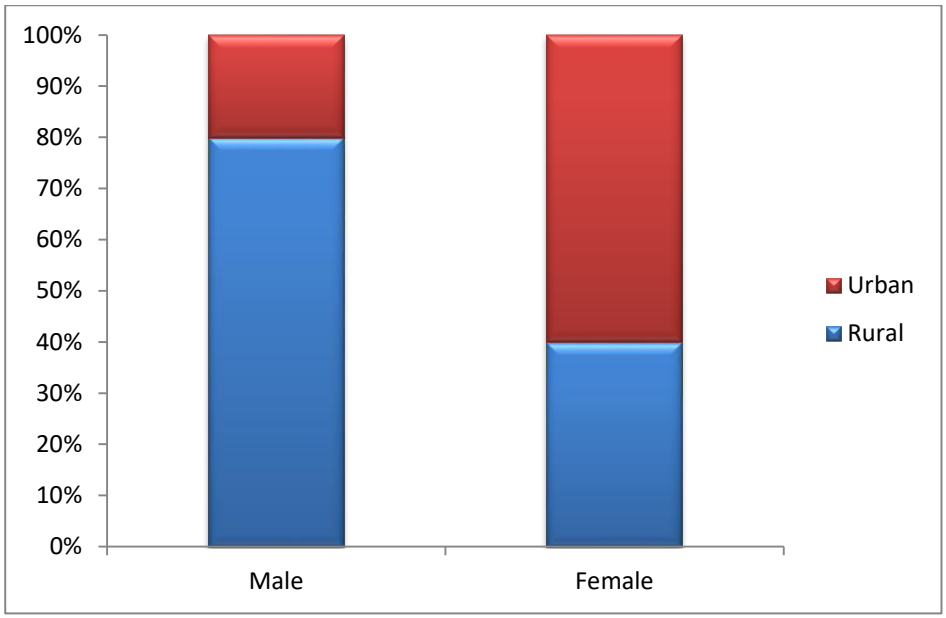


Lower socio economic status constituted above 32 (53.3%) of cases. Among this males 30(60%) and females 2(20%). Moderate socio economic status next group 26 (43.3%). Among this males 18 (36%) Females 8(80%). High socio economic status 2(3.3%) among this only males 2(4%). Among males majority belong to low socio economic status to 30(60%). Least belong to high socio economic status 2 (4%). Among female majority belonged to moderate socio economic status 8(80%) and no cases in High socio economic Status Group.

Table - 4

Residence

Place of residence	Male		Female		Total	
	N	%	N	%	N	%
Rural	40	80	4	40	44	73.3
Urban	10	20	6	60	16	26.7
Total	50	100	10	100	60	100

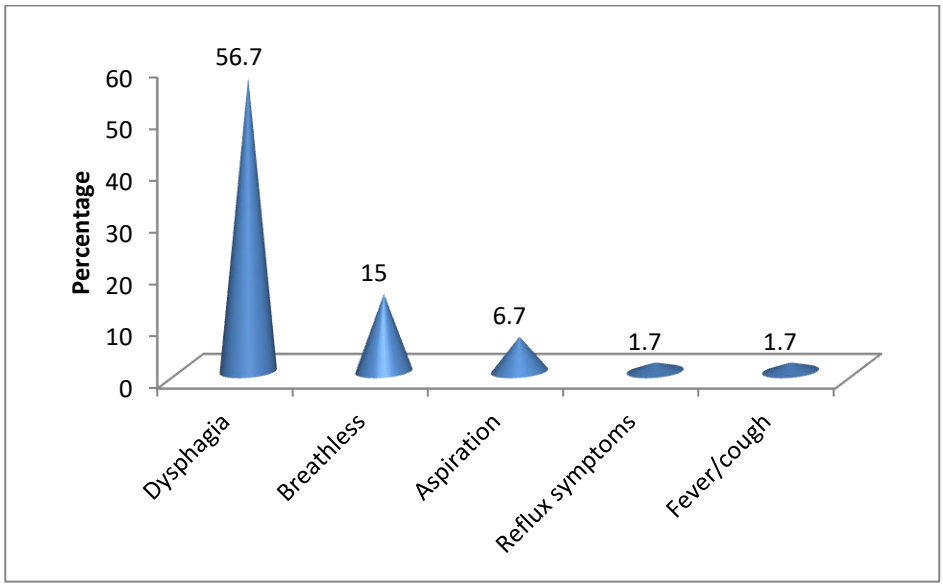


Maximum cases i.e. 44 cases (73.3%) were from rural area and minimum cases i.e. 16 cases (26.7%) were from urban area.

Table – 5

Clinical Presentation of patients with hoarseness of voice

	Frequency	Percent
Dysphagia	34	56.7
Breathlessness	9	15
Aspiration	4	6.7
Reflux symptoms	1	1.7
Fever/cough	1	1.7

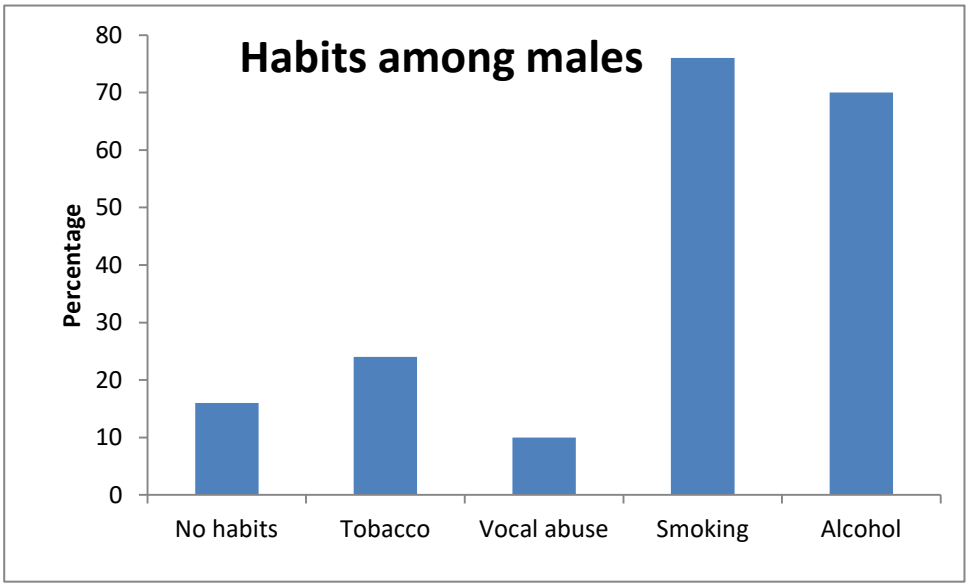


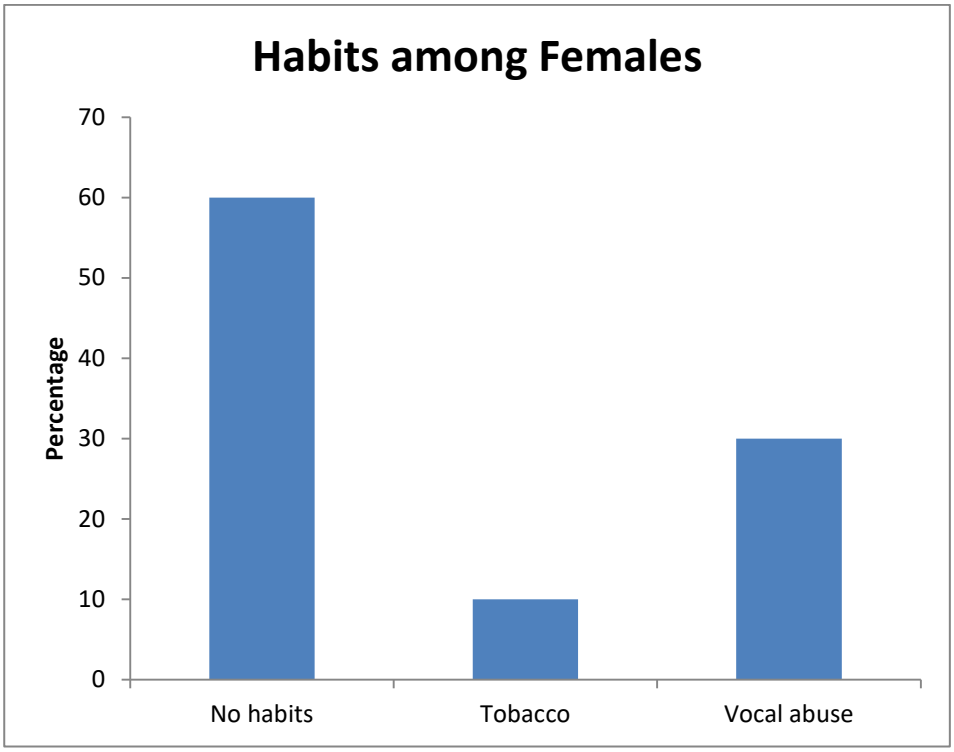
All 60 cases (100%) presented with hoarseness of voice. Most common associated complaint was dysphagia 34 cases (56.7%) and the least associated complaints were reflux symptoms and fever /cough 1 each (1.7%)

Table – 6

Habits

Habits	Male		Female		Total	
	N	%	N	%	N	%
No habits	8	16	6	60	14	23.3
Tobacco	12	24	1	10	13	21.7
Vocal abuse	5	10	3	30	8	13.3
Smoking	38	76	0	0	38	63.3
Alcohol	35	70	0	0	35	58.3



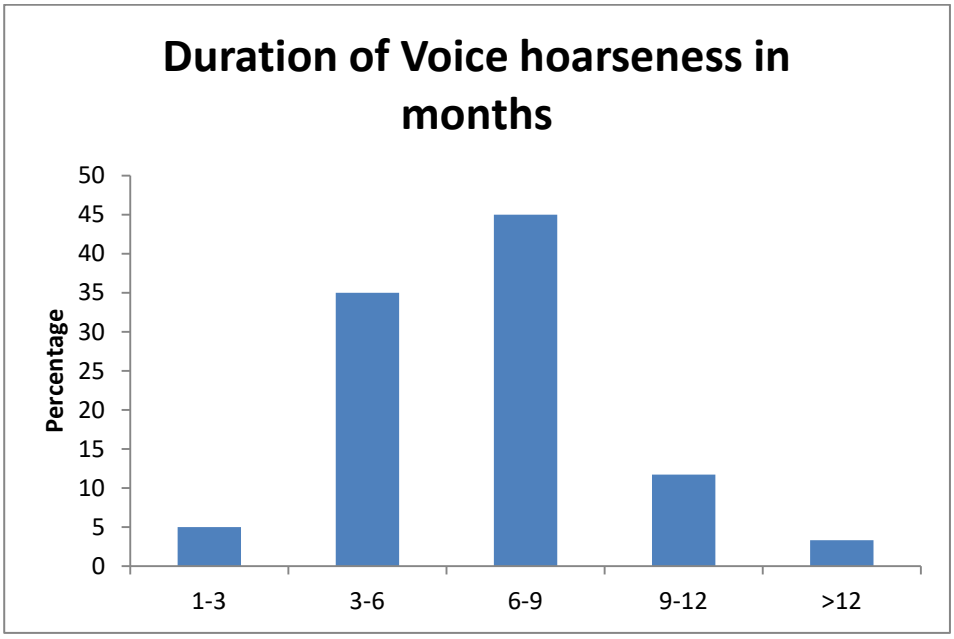


Commonest habit noted was smoking in 38 cases (63.3%) and least habit was vocal abuse i.e.8 cases (13.3%). Among males commonest habit was smoking i.e. 38 cases (76%) and least habit was vocal abuse, i.e. 5 cases (10 %).Among females maximum number had no habits group 6 cases (60%) and least habit was chewing tobacco preparation i.e. 1 cases (10%).

Table – 7

Duration of hoarseness of voice

Duration of Voice hoarseness in months	Frequency	Percentage
1-3	3	5.0
3-6	21	35.0
6-9	27	45.0
9-12	7	11.7
>12	2	3.3
Total	60	100.0

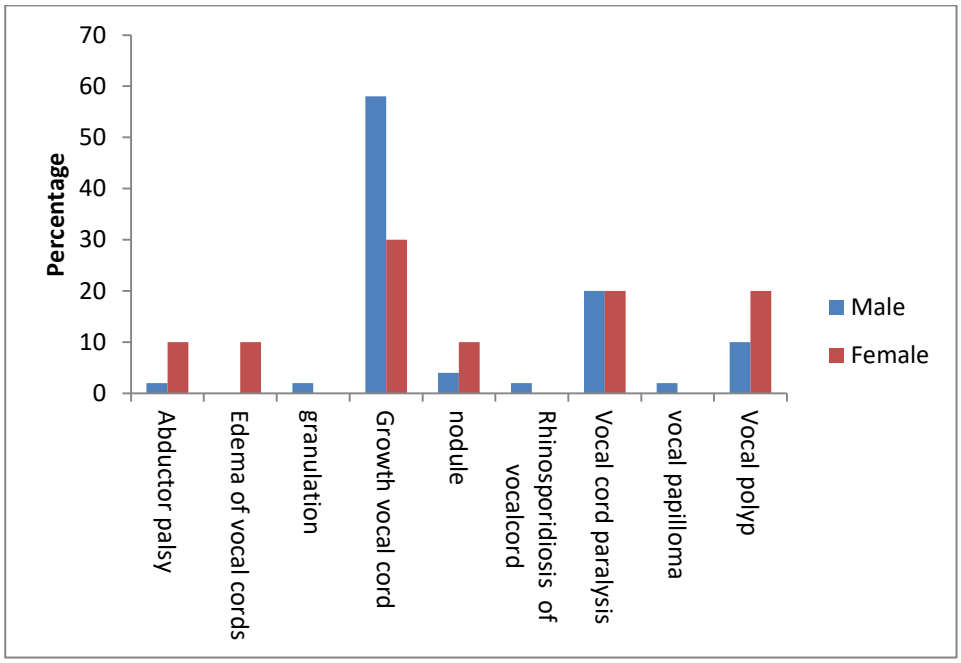


Maximum number of patients i.e. 27(45%) each presented during 6-9 month of duration. Least number of patients i.e. 2 case (3.3%) each presented during >12 month duration.

Table – 8

Indirect laryngoscopic (IDL) examination

Indirect	Male		Female		Total	
	N	%	N	%	N	%
Abductor palsy	1	2.0	1	10.0	2	3.3
Edema of vocal cords	0	.0	1	10.0	1	1.7
granulation	1	2.0	0	.0	1	1.7
Growth vocal cord	29	58.0	3	30.0	32	53.3
nodule	2	4.0	1	10.0	3	5.0
Rhinosporidiosis of vocalcord	1	2.0	0	.0	1	1.7
Vocal cord paralysis	10	20.0	2	20.0	12	20.0
vocal papilloma	1	2.0	0	.0	1	1.7
Vocal polyp	5	10.0	2	20.0	7	11.7
Total	50	100.0	10	100.0	60	100.0

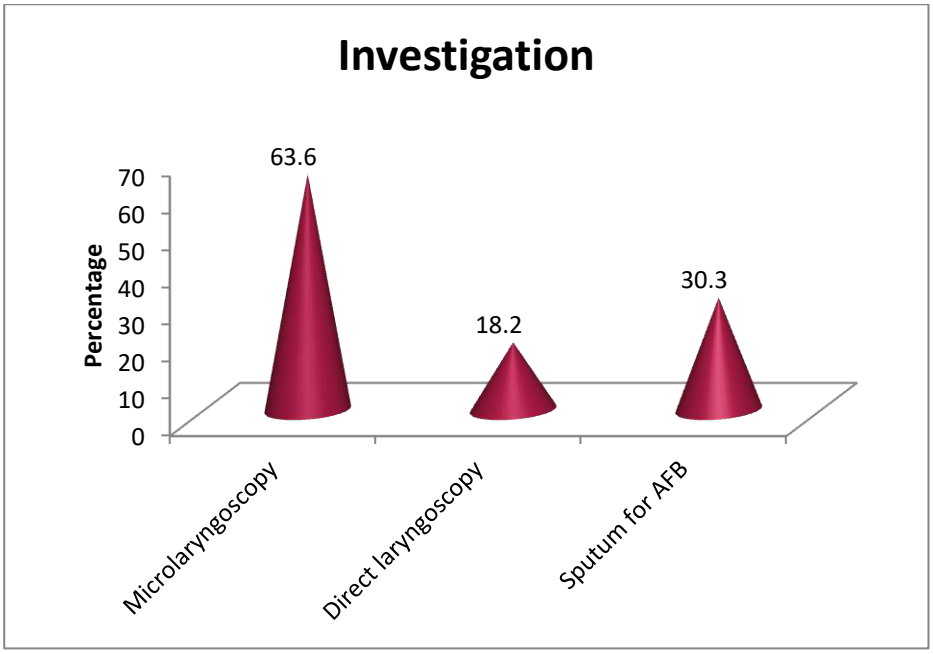


Maximum number of cases i.e. 32 (53.3%) presented with ulcero- proliferative growth of larynx and least number of cases, 1 case (1.7%) each showed granulation in laryngeal trauma, rhinosporidiosis, chronic laryngitis and vocal cord papilloma.

Table – 9

Investigations

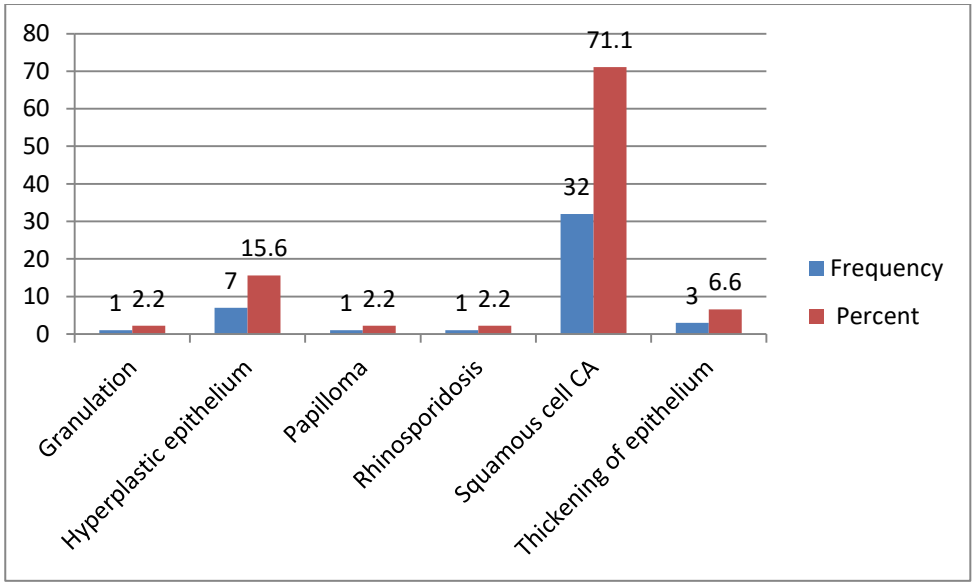
Investigation	Frequency	Valid Percent
Microlaryngoscopy	21	63.6
Direct laryngoscopy	6	18.2
Sputum for AFB	10	30.3



21 cases (63.6%) underwent microlaryngoscopic examination and 6 cases (18.2%) underwent dirctlaryngoscopic examination and 10 cases (30.3%) sputum for AFB tests.

Table 10-histopathology

Histopathology	Frequency	Percent
Granulation	1	2.2
Hyperplastic epithelium	7	15.6
Papilloma	1	2.2
Rhinosporidosis	1	2.2
Squamous cell Ca	32	71.1
Thickening of epithelium	3	6.6

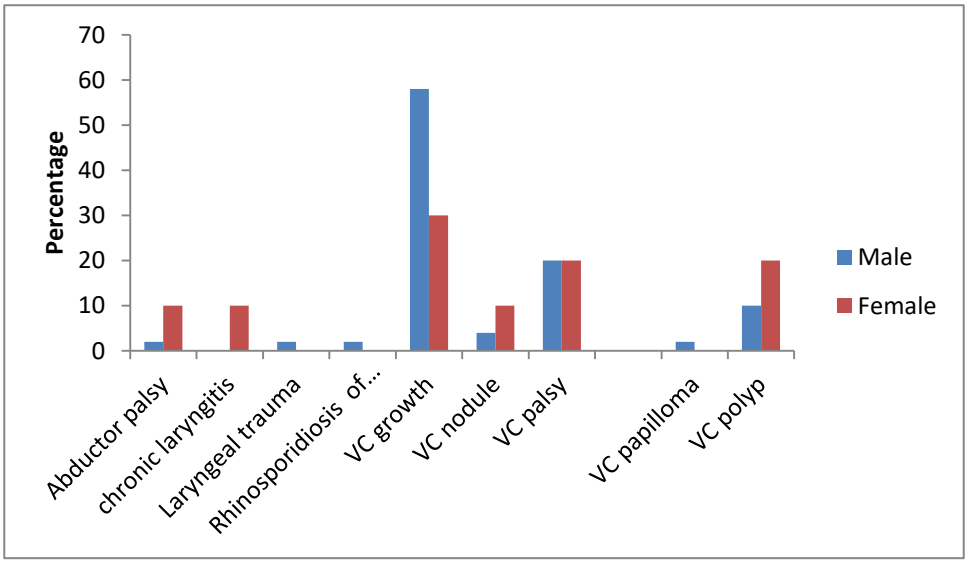


Out of 60 cases, only 45 cases were subjected to histopathological examination. Squamous cell carcinoma was diagnosed in 32 cases (71.1%). Granulation, rhinosporidisos, papilloma each one case (2.2 %). Hyperplastic epithelium 7 cases (15.6%). Thickening of epithelium with inflammation 3 cases (6.6%).

Table – 11

Aetiology of hoarseness of voice

Diagnosis	Male		Female		Total	
	N	%	N	%	N	%
Abductor palsy	1	2.0	1	10.0	2	3.3
chronic laryngitis	0	.0	1	10.0	1	1.7
Laryngeal trauma	1	2.0	0	.0	1	1.7
Rhinosporidiosis of vocalcord	1	2.0	0	.0	1	1.7
VC growth	29	58.0	3	30.0	32	53.3
VC nodule	2	4.0	1	10.0	3	5.0
VC palsy	10	20.0	2	20.0	12	20
VC papilloma	1	2.0	0	.0	1	1.7
VC polyp	5	10.0	2	20.0	7	11.7
Total	50	100.0	10	100.0	60	100.0

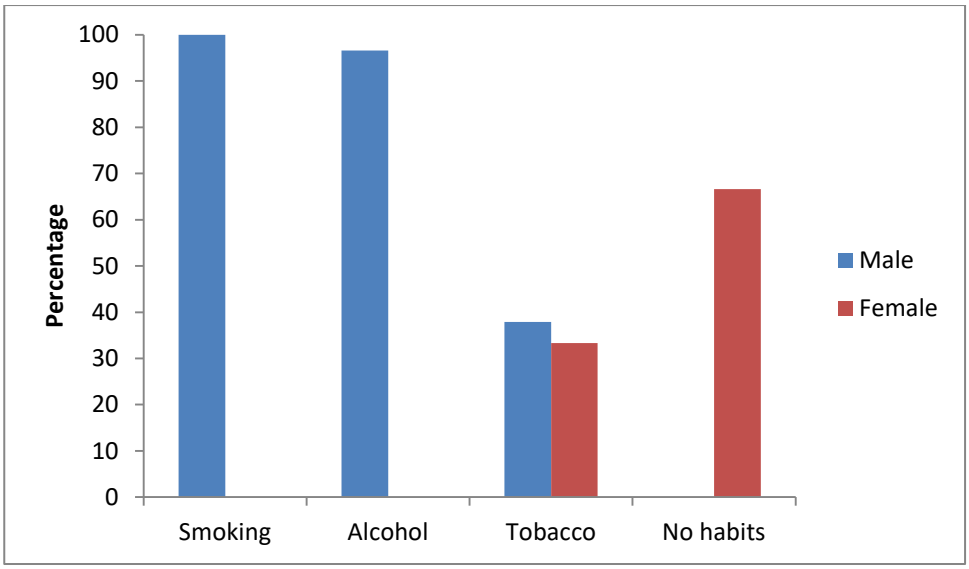


Commonest aetiology noted was malignancy comprising 32 cases (53.3%) and least were, 1 case (1.7%) Chronic laryngitis, laryngealtrauma, rhinosporidiosis, Papilloma. Among males commonest aetiology was malignancy i.e. 29 cases (58%). Among females commonest aetiology was malignancy 3 cases (30%).

Table – 12

Habits in laryngeal malignancy

Habits	Male(n=29)		Female(n=3)		Total(n=32)	
	N	%	N	%	N	%
Smoking	29	100	0	0	29	90.60
Alcohol	28	96.55	0	0	28	87.50
Tobacco	11	37.90	1	33.30	12	37.50
No habits	0	0	2	66.60	2	6.25



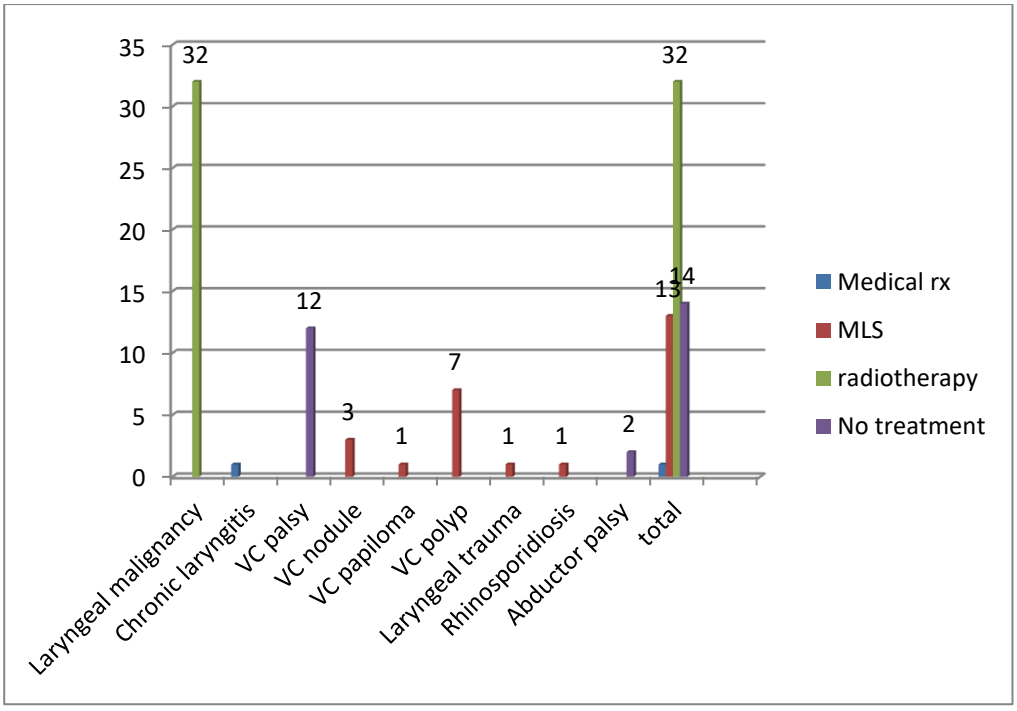
In 32 cases of malignancies smoking was noted in 29 cases (90.6%) and consumption of alcohol was noted in 28 cases (87.5%) and chewing tobacco preparations in 12 cases (37.5%) and no habit in 2 cases (6.25%).

Among males smoking was noted in all 29 cases (100%) consumption of alcohol was noted in 28 cases (96.55%) and chewing tobacco preparation noted in 11 cases (37.9%). Females maximum had no habits 2(66.6%) and 1 case (33.3%) tobacco chewer.

Table – 13

Treatment Given

diagnosis	Medical rx	MLS	radiotherapy	No treatment	total	
					N	%
Laryngeal malignancy			32		32	53.3
Chronic laryngitis	1				1	1.66
VC palsy				12	12	20
VC nodule		3			3	5
VC papiloma		1			1	1.66
VC polyp		7			7	11.8
Laryngeal trauma		1			1	1.66
Rhinosporidiosis		1			1	1.66
Abductor palsy				2	2	3.30
total	1	13	32	14	60	100
pecentage	1.67	21.67	53.33	23.33	100	100



Out of 60 cases, 32 cases (53.3%) were subjected for radiotherapy, 13 cases (21.78%) subjected for MLS. No Treatment was given for 14 cases (23.3%). Medical Treatment given for 1 case (1.7%). 32 cases of malignancy of larynx and

were subjected to radiotherapy . Besides radiotherapy these patients underwent tracheostomy due to respiratory distress and supportive treatment like analgesics for pain were prescribed. 1 case of chronic laryngitis underwent medical treatment that included antibiotics, anti-inflammatory, analgesics, decongestant, steam inhalation along with voice rest and speech therapy.

For 12 patients of vocal cord paralysis, no treatment was given. Among 3 patients of vocal nodule underwent MLS excision. 1 patient of papilloma, 7 cases of polyp and 1 case of granulation, papilloma underwent MLS excision followed by voice rest, speech therapy and medications.

Table – 14

Results of follow up

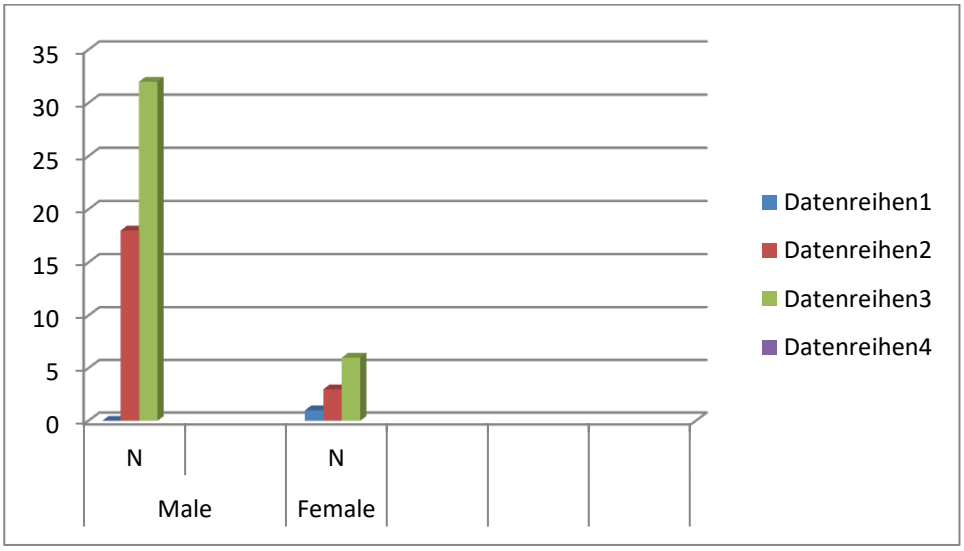
Diagnosis	Response						Total	
	Improved		No change		Worsened			
	N	%	N	%	N	%	N	%
Abductor palsy	0	.0	2	100.0	0	.0	2	100.0
chronic laryngitis	1	100.0	0	.0	0	.0	1	100.0
Laryngeal trauma	1	100.0	0	.0	0	.0	1	100.0
Rhinosporidiosis of vocalcord	1	100.0	0	.0	0	.0	1	100.0
VC growth	0	.0	0	.0	32	100.0	32	100.0
VC nodule	3	100.0	0	.0	0	.0	3	100.0
VC palsy	2	16.67	10	83.33	0	.0	12	100.0
VC papilloma	1	100.0	0	.0	0	.0	1	100.0
VC polyp	7	100.0	0	.0	0	.0	7	100.0
Total	16	26.7	12	20.0	32	53.3	60	100.0

Among 60 cases 16 cases (26.7%) showed improvement in voice, 32 cases (53.3%) showed worsening of voice and 12 cases (20%) had no change in voice on follow up.

Tble-15

Vocal professional level

LEVELS	Male		Female		Total	
	N	%	N	%	N	%
I	0	0	0	0	0	0
II	0	0	1	10	1	1.7
III	18	36	3	30	21	35
IV	32	64	6	60	38	63.3
Total	50	100	10	100	60	100



Koufman and isaacson (1991) classification level I (series)- Elite vocal performers (singers and actors)

Level II : Professional voice users (clergyman, politician, Public Speakers, Lecturer)

Level III : Non vocal Professional (Teachers, Lawyers)

Level IV : Non Vocal / Non Professionals (Labourers, Home makers, Clerk)

In the present study according to above classification there were no case in level I group. 1 case (1.7%) Level II. Level III. 21 cases (35%). Maximum number of cases belong to Level IV group 38 cases (63.3%).

DISCUSSION

Hoarseness is a symptom, not a diagnosis. It is one of the commonest symptoms and is invariably the earliest manifestation of a large variety of condition affecting voice apparatus. Parik[12] mentions that it is however strange that hoarseness as a subject has not attracted the attention of many workers. Hoarseness lasting longer than two weeks needs complete evaluation to rule out malignancy as a cause.

A total 60 cases of hoarseness of voice were studied during study period. In our study period incidence of hoarseness voice was 0.27% while in a study by Sambu Baitha[92] incidence of hoarseness was 0.32%, which nearly coincides with out study. In another study by Prabhu Khavasi[97] incidence of hoarseness voice was 0.3% which almost coincides with our study.

Age and Sex

In our study age of patients with hoarseness of voice ranged from 5 yrs to 80 yrs majority of patients i.e. 18 cases (30%) were in the group of 51-60 years and minimum number of patients i.e. 1 case (1.7%) was in the age of group of 11-20 years. In a study by Sambu Baitha[92] majority of patients i.e. 31 cases (28.18%) were in the age group of 31-40 years. In a study by Swapan Ghosh [93] maximum patients i.e. 28 cases (28%) were in the age group of 21-30 years. In study by Prabhu Khavasi[97] majority of patients 19 cases (27.14%) were in age group 41-50 yrs. Minimum number 1 case (1.43%) in the age group < 10 yrs.

In our study 50 case were males (83.3%) and 10 cases (16.7%) were females. Male to female ratio was 5: 1 which exactly coincides with other studies showing male preponderance. In study by Prabhu Khavasi[97] 48 cases (68.57%) were males and 22 cases (31.435) were females, thus male to female ratio of approximately 2:1 was observed. study by Sambu Baitha ,[92] where males 74 cases (67.27%) and female 36 cases (32.72%) were noted and also in study by Parikh[12] where males presented 67% and females 33% with male to female ratio of 2:1.

Occupation

In our study out of 60 case majority 35 (58.3%) belonged to labour class and least belong to farmer group 1 case (1.7%)

In study by Prabhu Khavasi majority of patients i.e. 34 cases (48.57%) were of labourer class and least i.e. 3 cases (4.29%) were in others group. In a study by Sambu Baitha et al[92] the same observation was made i.e. majority of patients were of labourer class (36.36%). In study by Swapan Ghosh [93] majority of patients were house wife (29%). In our study among males labour class predominated 33 (66%). Among females Housewife's predominated 5 (50%).

Socio economic status

In study by Prabhu Khavasi, majority of patients i.e. 34 cases (48.57%) belonged to low socio economic status and also majority of patients among males, i.e. 24 cases (50%) and females i.e. 10 cases (45.45%) belong to lower socio-economic status. In our study low SES constituted about 32 cases 53.3%) and also among this majority was males 30 (60%) and females 2 (20%). Low SES percent tage almost coincided with above study. Next group was with moderate SES 26

(43.3%) last was high SES 2 (3.3%).

Residence

In our study maximum number of \cases 44 (73.3%0 belong to rural area and mimimum number of cases 16 (26.7%) from urban area . In study by Prabhu Khavasi majority of patients i.e. 45 cases (64.29%) were from rural area and minimum cases, 25 cases (35.71%) were from urban area. Also in study by Sambu Bhaita [92] patients were predominantly from rural areas comprising of 83 cases (75.5%).

Our study coincides with above studies concluding maximum number of cases came from rural area.

Habits

Brock has mentioned inhaled irritants especially cigarette smoke as most important predisposing factors for hoarseness.[40] In study by Prabhu Khavasi [97]commonest habit noted was smoking i.e. 38 cases (79.12%) and vocal abuse (18.57%) was least common habit. In study by Swapan Ghosh[93] vocal abuse was noted in 72% of cases, and in study by Sambu Baitha [94] smoking was noted in 25.45% of cases, chewing tobacco preparation was noted in 17.27% and Alcohol in 12.72%. Parik[12] has found it in 20% of cases only and vocal abuse was 56%. In our study commonest habit / Predisposing factor noted was smoking 38 cases (63.3%) and leastwas vocal abuse 8 case (13.3%). This coincides with study by Prabhu Khavasi[97]. In our study majority of males were smokers 38 (76%) and among female majority had no habits. 6 cases (60%).

Clinical presentation

In our study hoarseness of voice seen on all 60 cases. Most common associated complaint was dysphagia 34 (56.7%) and least associated were reflux symptoms and fever/ cough 1 case (1.7%) each. In study by Prabhu Khavasi[97] hoarseness was noted in all the 70 cases (100%) and next common symptom was cough (42.86%), dysphagia 19 cases (27.14%) other symptoms were breathlessness, (15.17%), foreign body sensation in throat 10 cases (14.29%). In study by Sambu Baitha [92] hoarseness was observed in all cases (100%) least common symptom was noisy respiration (0.99%).

Duration of hoarseness of voice

In our study maximum number of patients 27 (45%) presented during 6-9 months duration. Least 2 cases (3.3%) during more than 12 month duration.

Study by Prabhu Khavasi[97] duration ranged from 3weeks to 5 years and maximum number of patients i.e. 20cases (28.57%) each presented during 1-3 months and 3-6 months of duration of hoarseness,

Indirect laryngoscopic (IDL) examination

In our study on IDL 32 cases (53.3%) growth was noted in the vocal cord. About 1.7% of cases each constituted granulation in laryngeal trauma, papilloma rhinosporidiosis and edema of vocal cord in laryngitis.

Study by Prabhu Khavasi[97] indirect laryngoscopic examination (IDL) commonest finding was – Ulceroproliferative growth involving larynx, 28 cases (40%). Vocal folds cyst and submucosal hemorrhage of vocal folds and false

cords were noted in 1.43%. In a study by Sambu Baitha [92], congestion of vocal cords noted in 34.54%, growth in only 9% of cases on IDL examination.

Investigations

In our study 21 cases (63.6%) underwent microlaryngoscopy and 6 cases (18.2%) direct laryngoscopic and 10 cases (30.3%) sputum for AFB. Prabhu Khavasi[97] While studying, 23 cases (32.86%) were subjected for direct laryngoscopic examination and 12 cases (17.14%) for microlaryngoscopic examination. In study byParikh[12] 60% of patient underwent microlaryngoscopy, in study by Sambu Baitha[92] DLS/MLS was done is 40 cases (36.36%), Sputum for AFB was done in 4 cases (5.71%) in our study.

Histopathology

In our study out of 60 cases, 45 cases were subjected to Histopathological examination. 32 cases (71.1%) showed squamous cell carcinoma. Hyperplastic epithelium 7 cases (15.6%). Thickening of epithelium with inflammation 3 cases (6.6%) and 1 cases 2.2%) each with granulation rhinosporidiosis, papilloma.

Prabhu Khavasi[97] study, biopsy was done in 34 cases (48.57%) and histopathological finding most commonly encountered was squamous cell carcinoma, in 28 cases (82.35%) and least common finding was subepithelial hyalinization, in 1 case (2.94%).

Aetiology of hoarseness of voice

In our study commonest aetiology observed was, malignancy of larynx and laryngopharynx 32 cases (53.3%). Among males commonest etiology was malignancy of larynx and 29 cases (58%) and among females malignancy of larynx and was only 30% (3 cases). 9 Among patients with malignancy males

were common 29 cases and females 3 cases with male to female ratio as 9:1 In study by Sambu Bhaita[92] incidence of malignancy was 14.54% with male to female ratio as 15:1. In study by Kadambari[13] incidence of malignancy was 18% and in study by Swapan Ghosh[93] incidence of malignancy was only 8% and in Parikh[12] incidence of malignancy was 12%.

In our study next common aetiology was vocal cord palsy 12 cases (20%). Among females it was common etiology 2 cases, (20%). And males 10 cases (20%) and male to female ratio was found to be 5:1. In study by parikh [12] and Kadambari[13] and Sambu baitha[92] tt was 3%, 9%,9% with male to female ratio in Sambu Baitha[92] was 9:1. Among females in our study second common etiology also included vocal cord polyp 2 cases (20%).

Third common aetiology was vocal cord polyp 7 cases (11.

7%). Among male incidence was 10% (5 cases) and among female 20% (2 cases), with male to female ratio as 2.5:1. In study by Swapan Ghosh[93], Parikh[12] and Sambu baitha [92] incidence of vocal cord polyp were 23%, 15%,4.54% with male to female ratio 3.6 :1 in Swapan Gosh [93] and 1:1.5 in Parikh study.

The fourth common aetiology was vocal cord nodules 3 cases (5%) with male to female ration 2:1 Among males 4% (2 cases) were affected and among females 10% (1 cases) patients had vocal nodules. Vocal nodules were the commonest aetiology in study by Parikh[12] (50%) with males 43.3% and females 56.7% and also study by Swapan Ghosh[93] it was commonest etiology with incidence of 30% with male to female ratio 1:1.5. In study by Sambu Baitha[92] incidence was only 12.72% with male to female ratio 1:1.3. The fifth common

etiology was bilateral abductor palsy 2 cases (3.3%), All the patients were male and female 1 each. Male to female ratio 1:1. Vocal cord papilloma was found in 1 cases (1.7%) male. to female ratio as 1:2 as one male and 2 female patients presented. Chronic Laryngitis, Laryngeal trauma, rhinosporidiosis 1 case each (1.7%).

Association of Habits in malignancies

In our study, among malignancies 32 cases (53.3%), smoking was noted in 29 cases (90.6%) and consumption of alcohol 28 cases (87.5%) and chewing tobacco preparation in 12cases (37.5%).Among males smoking was noted 100% and alcohol consumption 96.5% and chewing tobacco preparation in 11 cases (37.9). Among females 1 case 33.3%) Tobacco chewing was present. Other females had no specific habits.

In study by Sambu Baitha et al.[94] Smoking was noted in 75% of cases epidemiological data has demonstrated a strong correlation between tobacco use and laryngeal cancer.[94] Sokic et al 1995, studied risk factors for laryngeal cancer and noted association of smoking and alcohol.[86]

Treatment given

Among 60 cases 32 cases (53.3%) were treated by radiotherapy. MLS-excision of lesion for 13 cases (217%). No treatment for 14 cases (23.3%) and medical treatment for 1 case (1.7%) . commonest condition was Laryngeal carcinoma 32 (53.3%) treated by Radio Therapy and vocal cord polyp was commonest condition treated by MLS 7(11.8%). In study by Swapn Gosh (83%) treated by MLS.

Results on follow up

Out of 60 case in our study improvement of voice was shownby 16 cases (26.7%), worsening of voice seen in 32 cases (53.3 %) and no change 12 cases (20%) on follow up.

Most common condition which improved with treatment was vocal cord polyp 7 cases showing about 10% improvement. study by Swapan Gosh[93] also showed 100% improvement in vocal cord polyp treatment. Patients with malignancy were subjected to radio therapy and all cases voice worsened 32(100%). In case of vocal cord palsy 12 cases 2 case improved 16.6%) on follow up without treatment. 10 cases 83.3% no change of voice was noticed.

2. cases of abductor palsy showed no change in voice on follow up without treatment.

Cases of vocal papilloma treated with MLS shown 100% improvement. In study by Swapan Gosh[93] improvement was 70%.

Case of vocal cord nodule 3 cases 100%) improvement in voice was notedbut study by Swapan Gosh[93] showed only 93% improvement.

1 case of rhinosporidiosis and 1 case Laryngeal Trauma improvement was 100% with MLS. 1 case of Chronic laryngitis treated medically showed 100% improvement.

Vocal Professional Level

Koufman and Isaacson (1991) evolved a classification of vocal professionals based on their voice use and risk[96]

Level 1 : (Elite vocal performer)

Included sophisticated voice users like singers and actors, where even a slight vocal difficulty cause serious consequences to them and their careers.

Level 2 : (Professional Voice users)

Even moderate vocal difficulty would hamper adequate job performance. Example politician, clergyman, public speaker, Lecturer.

Level 3 : (Non vocal Professional)

It includes Teachers and Lawyers. They perform their jobs with slight or moderate voice problem.

Level 4 : (Non vocal/ Non Professional)

Includes labourers, homemakers and clerk. These are persons who are not impeded from doing their work when they experience any kind of dysphonia.

In our study NO CASES were reported in level 1 group. 1 cases 1.7 % was reported in level 2. 21 cases 35% in level 3 group. Maximum number of cases belong to level 4 38 cases 63.3%.

In study conducted by Hansabanjara,[1] Varsha, Digvijaysingh and Anuj Gupta Level 1 group had 1.59% cases Level 2 had 3.59 % and level 3 9.56% and 4, 85.26% cases. Batra et al (2004) found 52.9% of patient in level 4. In our study Level 4 is the major group which coincides with above mention studies.

CONCLUSION

Production of voice is complex mechanism. The vocal folds produce tone that becomes modified by pharynx, palate, tongue, nose and lips to generate the individual sounds of speech.

Not much work has been carried out in this field, Parik[12] (1991) mentions, and "It is however strange that hoarseness as a subject has not attracted the attention of many workers" In our present study incidence of hoarseness voice was 0.27% of total ENT cases.

Maximum number of cases18 (30%) were in the age group of 51-60 years, among males common age groups was 51-60 years (32%) and among females 31-40 years of age group (40%).

Hoarseness was commonly found in labourer class 35 (58.3%). Among males this was commonest group comprising 33 (66%) and among females commonest was housewife category 5 (50%). Lower socio economic group was commonly noted among patients (53.3%), also in males (60%) and females (20%). Female majority belonged. To Moderate SES 8 (80%)

Majority patients were from rural area 44 (73.3%).

Smoking was commonly encountered habit among males 38 (63.3%). Among females, majority had no habit 60%. Least habit was vocal abuse 5 (10%).

Along with hoarseness (100%) other symptom with which patient presented were dysphagia 34 (56.7%), and least associated complaints was reflux symptoms & fever and cough each 1.7%

Maximum number of patients presented with hoarseness of voice with duration of 6-9 months about 27 cases (45%).

On indirect laryngoscopic examination commonest finding was laryngeal growth 32 (53.3%)

Microlaryngoscopic examinations and direct laryngoscopy 6 (18.2%) and sputum for AFB in 10 (30.3%).

Among 45 histopathological studies, commonest finding was squamous cell carcinoma 32 (71.1%).

Our study showed following aetiological factors for hoarseness of voice.

Laryngeal malignancies	:	53.3%
Chronic laryngitis	:	1.7%
Vocal cord paralysis	:	20%
Vocal cord nodules	:	5%
Vocal cord papilloma	:	1.7%
Vocal cord polyp	:	11.7%
Bilateral abductor palsy	:	3.3%
Laryngeal trauma	:	1.7%
Rhinosporidiosis of vocal cord	:	1.7%

Laryngeal malignancy was the commonest cause of hoarseness of voice (53.3%) and males were commonly affected (58%). Smoking was noted in all male patients with malignancy (100%), along with alcohol consumption in 96.55% and chewing tobacco preparation in 37.9% of cases.

Vocal cord Palsy was next common cause for hoarseness of voice (20%) and was found 20% among males and 20% among females, with male to female ratio 5:1.

No treatment was given for vocal cord paralysis (20%) cases.

Vocal cord palsy (20%) & bilateral abductor (3.3%) palsy. On follow up showed no change in case of abductor palsy2 (100%) & 10 cases (83.3%) of vocalcord palsy. But 2 cases (16.67%) of vocalcord Palsy Improved.

7 cases(11.7%)) of vocalcord polyp was found and it was common in males (10%).Vocalcord polyps were treated with MLS surgery and improvement noted in 100% of cases.

3 case of vocal nodule (5%) Improvement voice in vocal nodules was 100%.It was common among male patients(4%)with male to female ratio2:1.

Bilateral abductor palsy 3.3%,among this male 1(2%) and female 1(10%) with male to female ratio 1:1

1 cases of vocal fold papilloma (1.7%) were treated with MLS excision, improvement of voice present in (100%).Males were commonly affected about 2%.

1 case (1.7%) of laryngeal trauma was treated MLS & improvement was 100%.

1 case each 1.7% presented with chronic laryngitis and rhinosporidiosis. Chronic laryngitis (1.7%) was treated medically and there was 100% improvement in voice. Rhinosporidiosis was treated with MLS excision and 100% improvement in voice noted.

Patients who had laryngeal malignancies(53.3%) were subjected to radiotherapy and hoarseness worsened in all cases. Tracheostomy was done in 15 cases.

Among 60 cases 26.7% showed improvement of voice, (53.3%) showed worsening and 20% showed no change in voice after treatment.

Based on vocal professional level classification, majority 63.3% belonged to level IV category non vocal non Professional.

Our conclusion is middle aged and elderly patients presenting with hoarseness of voice of more than two-week duration along with habits of smoking alcohol consumption and chewing tobacco preparation, malignancy must be ruled out.

Appropriate treatment after proper diagnosis is key to treatment and if patient follows up regularly voice can be restored to near normal.

SUMMARY

Incidence of hoarseness of voice was observed to be 0.27% of all ENT OPD cases. Patient's age ranged from 5 yrs to 80 yrs and majority of patient were of 51-60 yrs age group. Male to female ratio was 5:1, Labourer constituted single large group (58.3%). Majority of patients were from lower socio-economic status (53.3%). Majority of patients were from rural area. (73.3%) Smoking was commonest habit (63.3%). Maximum patients presented with duration 6-9 (45%) months. Apart from hoarseness other Most Common symptom was dysphagia (56.7%). Ulceroproliferative growth was commonest finding on indirect laryngoscopic examination (53.3%). Squamous cell carcinoma was commonest histopathological finding (71.1%). Malignancy was the commonest etiological leading to hoarseness (53.3%). Among males common aetiology was malignancy (58%) and among females also Malignancy was commonest aetiology (30%). 1.66% cases were treated medically, 21.78% cases underwent microlaryngeal surgery and 53.3% of cases treated with radiotherapy, and no treatment was given for 23.3% of patients.

On follow up study about. 26.7% showed Improvement in voice 53.3% of cases showed worsening of voice and 20% of case there was no change in voice .

In case Laryngeal Malignancy, most common predisposing factor noted was smoking (90.6%) followed by alcohol (87.5%).

Vocal professional level classification Maximum belonged to Non vocal/Non Professional (level IV) group constituting 63.3%.

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LIST OF ABBREVIATIONS USED

1.	AFB	Acid fast bacilli
2.	AIDS	Acquired immunodeficiency syndrome
3.	DL	Direct laryngoscopy
4.	EGG	Electroglottography
5.	GER	Gastrooesophageal reflux
6.	IDL	Indirect laryngoscopy
7.	LPR	Laryngo pharyngeal reflux
8.	MLS	Microlaryngoscopy
9.	RRP	Recurrent respiratory paillomatosis
10.	TEP	Tracheo esophageal puncture
11.	VLS	Videolaryngostroboscopy
12.	HPV	Human papilloma virus
13.	VC	Vocal cord

STUDY PROFORMA

Patient particulars: Informant:

Serial No:

Name: Age: Sex:

OP/IP Number:

Address / Residence

Occupation

Socio Economic Status

History

Hoarseness Of voice & its duration

Cough

Dysphagia

Breathlessness

Aspiration

Reflux Symptoms

Personal History

Addiction/ habits	Voice abuse
	Smoking
	Alcohol
	Tobacco
Other History	History of Intubation
	History of previous laryngeal surgery
	History of trauma
	History of head & neck surgery

Local Examination

IDL

Growth

Congestion of vocal cords,arytenoids,epiglottis

Vocal cord palsy

Vocal cord nodule/ polyp/cyst

Vocal cord papilloma

Investigations

Direct laryngoscopy

Micro laryngoscopy

Sputum for AFB

Histopathology

Diagnosis

Management

Medical Management

Drugs

Voice rest

Speech therapy

Surgical Management

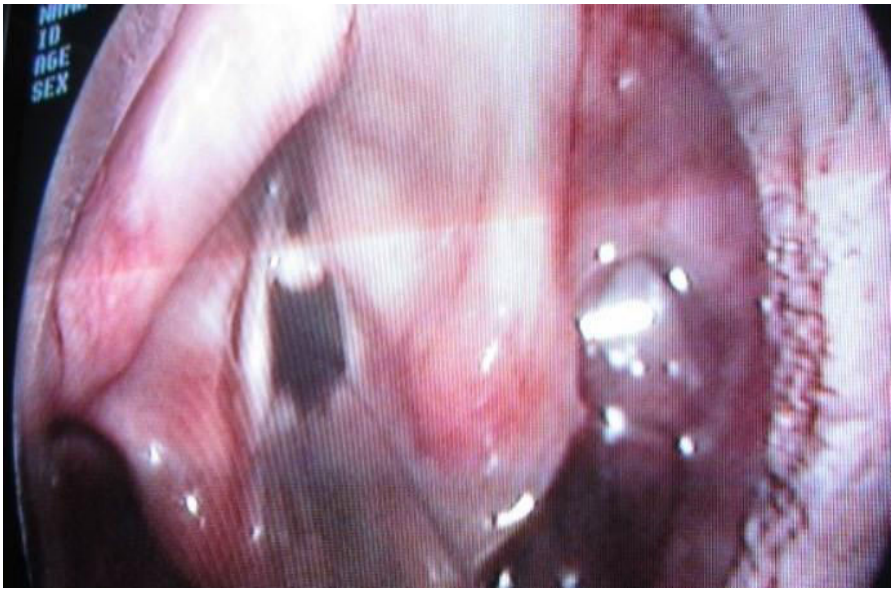
Microlaryngeal excision

Others

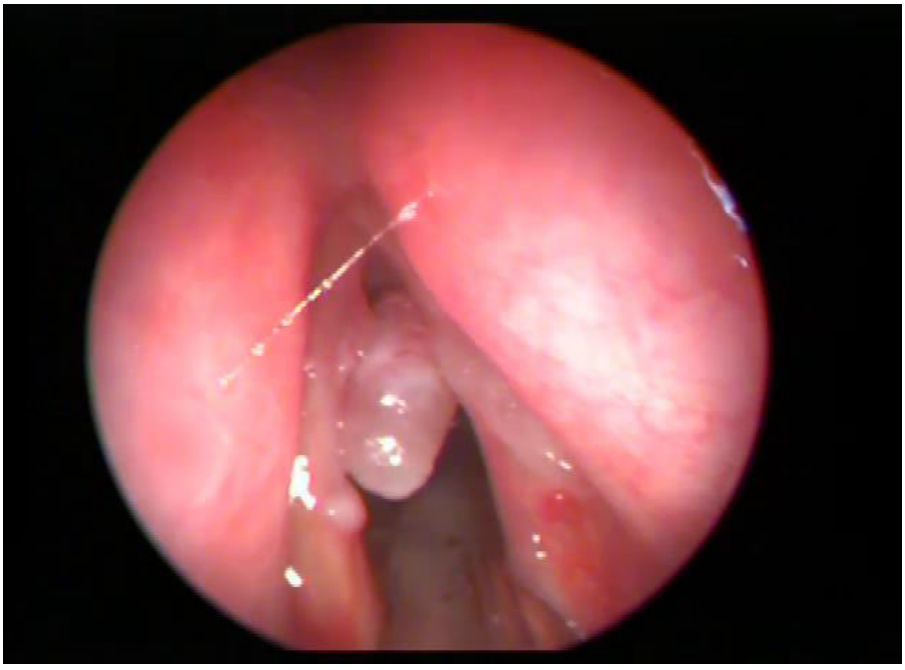
Radiotherapy

Follow up(after 3 months)

Voice - Improved,worsened,No change

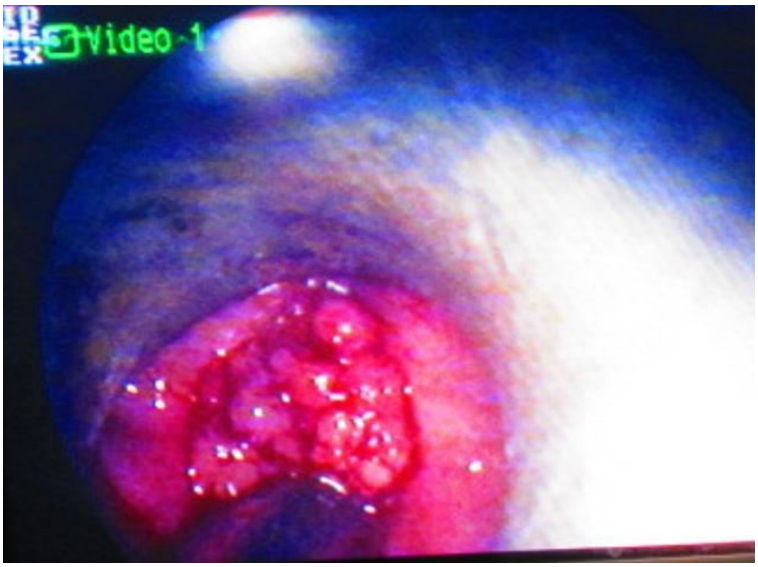


VC polyp

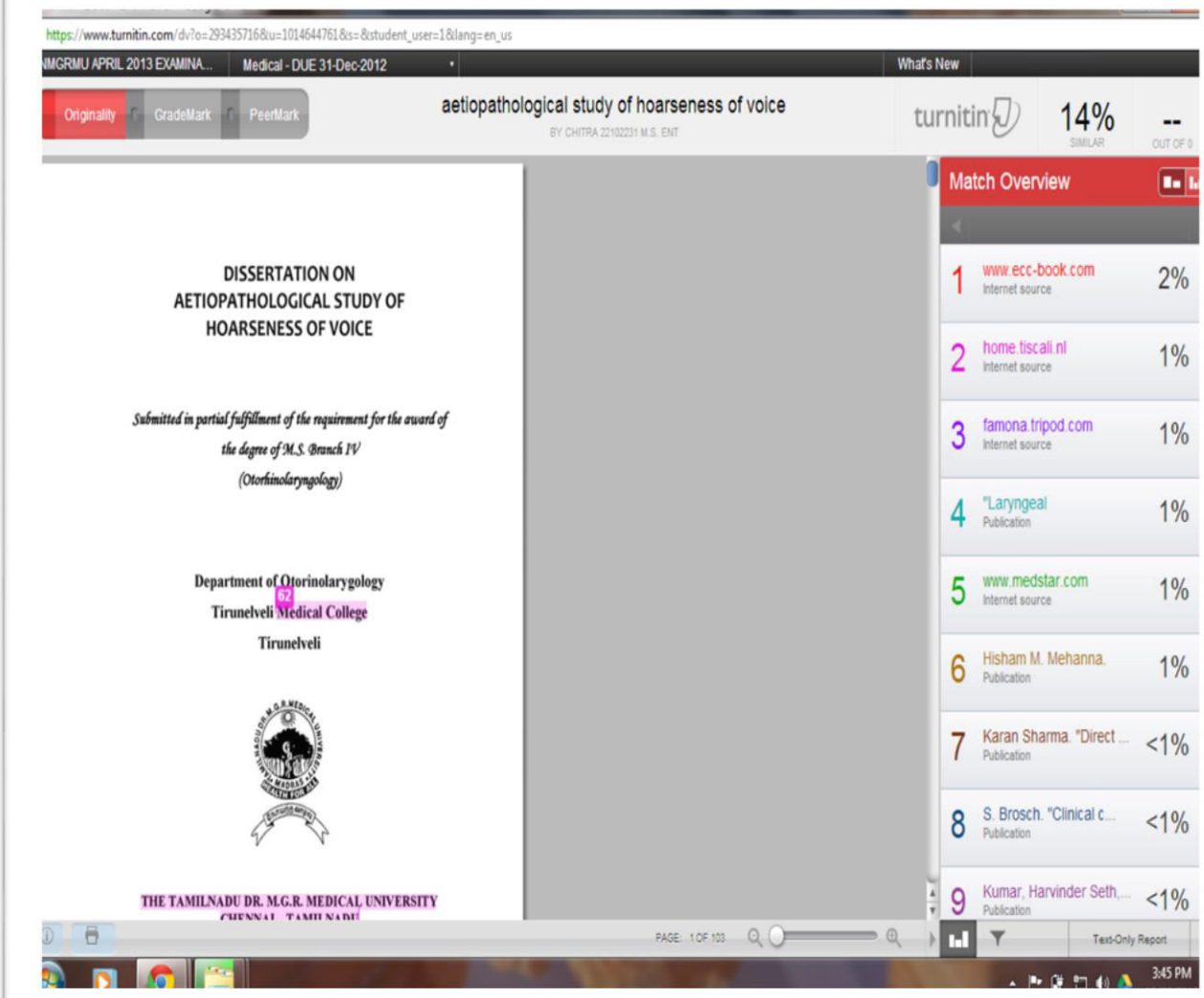


VC polyp

VC papilloma



VC papilloma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